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THE PERITONEUM

VOL. I

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THE PERITONEUM

VOL. I

STRUCTURE AND FUNCTION IN RELATION TO THE PRINCIPLES OF ABDOMINAL SURGERY

BY

ARTHUR E. HERTZLER, M.D., F.A.C.S.

SURGEON TO THE HALSTEAD HOSPITAL, HALSTEAD, KANSAS; ASSOCIATE PROFESSOR OF SURGERY, UNIVERSITY OF KANSAS; FORMERLY PROFESSOR OF PATHOLOGY, EXPERIMENTAL SURGERY, AND GYNECOLOGY, UNIVERSITY MEDICAL COLLEGE, KANSAS CITY, MO.

A CONTRIBUTION FROM THE LABORATORY OF THE HALSTEAD HOSPITAL, AND FROM THE DEPARTMENT OF ANATOMY OF THE UNIVERSITY OF ILLINOIS

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TO

AGNES, HELEN, AND MARGARET

MY DAUGHTERS

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PREFACE

This study was begun during my student days, now twenty-five years ago, and has occupied my leisure moments during all these years. Being done in conjunction with an active surgical practice, it naturally partakes of the practical viewpoint.

The first volume discusses the more abstract problems, while the second volume has to do with the practical aspects.

This being a research I have not felt obligated to treat all chapters equally and in the same way. In those chapters in which my own work is chiefly represented, there was in some instances little literature to consider. In others, for instance, that on Tuberculosis, where my own material was limited, the literature was reviewed extensively. On the whole, my reasons for the selection of the literature quoted are purely personal, and no attempt has been made to judge the permanency of the value of the papers selected for the bibliography.

Though I have done most of the work alone, much help was received from the frequent association with Dr. Montrose T. Burrows, Associate Professor of Pathology, Washington University Medical School; and in recent years Dr. Albert C. Eycleshymer, Professor of Anatomy, University of Illinois Medical School, has extended valuable aid and criticism.

I am particularly indebted to Mr. Tom Jones, the artist, for his limitless patience in reproducing specimens. Mrs. Rosa M. Hibbard, Librarian of the Kansas City Medical Library, has rendered valuable assistance in checking up the literature. The courtesy of the publishers has converted the usual task of seeing a work through the press into a real pleasure.

A. E. H.

Kansas City, Mo.

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THE PERITONEUM

VOL. I

CHAPTER I

PHYSIOLOGY OF THE PERITONEUM

I. **The Nature of the Peritoneum.**—The fundamental difficulty in the study of the peritoneum is the inability to formulate a clear idea of the function of this organ. The peritoneum from the morphologic point of view may be considered essentially a protective membrane. Its anatomic peculiarities are such as to best adapt it to the purpose of a protecting structure. The physiologic characteristics likewise subserve the same end. But for this membrane the movements of the abdominal viscera necessary to the performance of their function would be impossible. In this it resembles closely the synovial membrane of joints and tendons. It differs from these only in the viscidity of the fluid it produces and in the rapidity with which it responds to outside influences.

The ready response of the peritoneum to injury led to the assumption that its function had to do with the protection of the abdominal viscera from reactive processes. This idea was substantiated by the fact that the peritoneum responds quickly to diseases of subjacent organs. It is a mistake to assume that the peritoneum employs methods or agencies differing in character from the protective forces of other tissues of the body. This point will be fully discussed under pathology.

The older investigators regarded the peritoneal cavity as a huge lymph sac. This conception, it is important to remember, antedates the knowledge of the nature of the lymphatic circulation. We now know, thanks to the labors of Sabin, that the lymph channels consist of a closed system of vessels, closely allied to the blood

vessels, both in origin and structure. An examination of the peritoneal fluid shows no resemblance to the contents of the lymph vessels. Adler and Meltzer compare the lymph stream with the rectum, regarding it as an avenue by which waste products are carried off. There is no proof, they point out, that the character of the fluid in the connective-tissue spaces and in the lymph vessels is the same. The only resemblance is that both contain a clear fluid, poor in corpuscular elements. The function of the lymph vessels is to transport lymph, but obviously, the function of the peritoneum can not be to transport lymph.

By lymph spaces we mean clefts in the tissues outside of the lymph vessels. That these spaces have nothing to do with the lymph vessels Adler and Meltzer have attempted to emphasize by abandoning the term entirely, and applying the noncommittal term connective-tissue spaces.

When we compare the peritoneal cavity with the connective-tissue spaces it becomes at once apparent that a similarity does not exist. The connective-tissue spaces are the regions of tissue metabolism, where the reconstruction of tissues and the resorption of deleterious products of metabolism take place. That the peritoneal cavity is the scene of such activity will not be seriously maintained.

From the foregoing it becomes apparent that the peritoneum is a protective organ whose function is to provide a surface which will permit the organs which it covers to move upon each other without friction. The prominent part the peritoneum plays in the protection of the organism against accidents to the intestinal tube is due to the facility with which it responds to injury due to its abundant circulation.

The conception of the nature of the peritoneum as a protective organ in a purely anatomic sense does not absolve one from the necessity of a careful study of the processes of absorption and exudation. By these functions the peritoneum is enabled to maintain a proper degree of lubrication. Their exact balance is necessary in order that it may remain in a state of efficiency.

Much of the literature bearing on the physiology of the peritoneum was written on the assumption that the peritoneal cavity is a lymph space. The old writers believed absorption and lymph

production were identical. Since these studies bear a direct relation to the problem of absorption, in its present sense, they are worthy of review.

II. **The Formation of Lymph.**—Since the measure of lymph production was obtained by the determination of the amount of fluid that escaped from the thoracic or other large lymphatic ducts, it follows that in order that the fluid might escape it became necessary that the fluid should be derived from some source—the connective-tissue spaces or the peritoneal surfaces, as the case might be.

The fluid occupying the tissue spaces may be regarded as a part of the blood plasma which has escaped through the blood-vessel walls. The interstitial spaces is the laboratory of metabolic exchange. After performing the function of exchange of nutrition the residue escapes by passing through the walls of the lymph vessels, and is propelled through these channels.

It is fluid that has escaped from the blood vessels and has undergone changes incident to metabolism and has subsequently escaped into the lymph sewerage system that can properly be called lymph. Only such factors, therefore, as increase the intensity of metabolic activity can properly be said to augment the production of lymph. It will be noted that the means which were employed to increase the outflow from the lymph channels were factors which augment osmosis, but can not influence metabolism.

It must be noted at the onset that much confusion is caused by the change in the conception of what constitutes lymph. The formation of lymph, therefore, as here discussed has to do with the problem of absorption rather more than with the production of lymph.

The fundamental difficulty in discussing this problem arises from the inability to state our premises clearly. The whole literature reflects the uncertainty of the viewpoint of the investigator. At the outset it is necessary to guard carefully against the tendency to translate results of studies of one kind into general terms of another regarding the exact nature of which we are ignorant. For instance, we study the action of solutions on a dialyzing membrane, and at once conclude that absorption from the peritoneal cavity must be controlled by similar laws. We observe certain phenomena

when solutions of known character are introduced into the peritoneal cavity, and we at once assume that similar phenomena will take place with normal or pathologic fluids. Constant care, therefore, is necessary in order that our conclusions shall not be more inclusive than our minor premise.

With these preliminary remarks we may proceed to the examination of the studies having to do with the variation in the escape of fluid from the lymph trunks.

In general terms these studies may be divided into three general divisions. The first of these may be designated as Ludwig's theory and may be called the *filtration* theory. The general hypothesis underlying it was that increasing the intracapillary pressure would produce increase of fluid escaping through the lymphatics. This theory was followed by Heidenhain's which may be designated as the *secretion* theory. The third may be called the theory of *decreased resistance*.

1. *Ludwig's Theory.*—The first detailed study of this problem was made on the diaphragm by Ludwig and his pupils. These investigators regarded the lymph as a filtrate of blood plasma and believed its difference in composition was due to the fact that the blood capillaries offer a greater resistance to some substances than to others. Thus the salts in the blood plasma and in the tissue fluids being about equal according to this view, the capillary walls must offer little resistance to the passage of salts. Albuminates being less in amount in the tissue fluids than in the blood, the capillary walls must offer resistance to the passage of albumin. Brücke believed that the reason the fluid escaping from the lymph vessels contains less albumin than the blood was because some of the albumin was used up in its passage through the tissue spaces in the process of nutrition. We see here already a recognition of the relation of the production of lymph to the repair of tissue.

According to these older authors the amount of lymph produced was dependent on the degree of intracapillary pressure. Increase of intracapillary pressure was produced according to them by three factors; (1) Hindered venous outflow (Tomsa, Emminghaus, Sotnitschewsky); (2) Increased arterial inflow; (3) The overfilling of the vessels with blood.

2. *Heidenhain's Theory.*—Already Nasse had studied the influ-

ence of osmosis on lymph formation, and Rogowicz had noted that curare increased the flow of lymph without coincident changes in the circulation. It remained for Heidenhain to emphasize the weakness of Ludwig's theory and to present his own secretion theory.

Heidenhain reviewed Ludwig's studies by increasing the pressure in the aorta or by tying the portal vein, or both of these together and observed the result on the amount and character of the fluid flowing through the thoracic duct. By varying the pressure in the aorta he failed to find a corresponding variation in the flow of the lymph. The closure of the portal vein did, however, increase the amount of fluid flowing from the thoracic duct but decreased its protoplasmic contents and its coagulability. He demonstrated that even when the pressure in the aorta sank to zero there was still a flow of lymph for an hour or two. He concluded, therefore, that the lining cells of the vessels were the active factors.

The chief argument in favor of Heidenhain's theory was found in the action of certain substances, called "lymphagogues," on the production of lymph. These substances,—muscle extracts of cray fish, extracts of leeches, extracts of the intestines and liver of dogs, peptone and albumin,—increased the amount of lymph and lessened its coagulability. He interpreted this to mean that the vascular endothelium possessed a secretory power which was stimulated by the substances used. It is interesting to note that bacteria were observed to act in the same way. Heidenhain experimented with a second series of materials,—concentrated solution of crystalloid substances, sugar, urea and various salts. These produced an extraordinarily increased flow of lymph. He believed, for instance, that when sugar reaches the capillaries the cells lining these vessels seize on this sugar and excrete it into the connective-tissue spaces. After reaching these spaces it abstracts water from the vessels making the lymph spaces hydremic.

Hamburger concluded that the walls of the blood vessels possess the property of maintaining the hydroscopic properties of the plasma at a constant point. He demonstrated that the osmotic pressure of normal lymph is greater than that of corresponding blood serum. He ascribed this to the breaking down of molecules of high potential energy into small stable molecules of low potential energy in the process of metabolism.

3. *Theory of Decreased Capillary Resistance.*—Heidenhain's theories were soon actively attacked. Rogowicz in his studies with curare made the suggestion that the increased flow of lymph might be due to changes in the vessel walls. Starling showed first of all that the character of lymph varies according to the organ from which it was derived. The lymph from the liver shows a higher concentration than that derived from the portal vein. When the aorta is occluded the pressure falls but little in the liver, while in the region drained by the portal vein the fall is marked. The flow of lymph from the liver is unhindered, and, since this is the source of the more concentrated lymph the findings of Heidenhain are readily explained.

Starling proved by actual measurement of blood pressure that strong solutions of sugar and salt produced a marked flow of fluid from the tissues into the blood, producing an increased capillary pressure. This could be prevented by a preliminary lowering of blood pressure by bleeding, and no increase in lymph production results, while the stimulation to the vessel endothelium from the salt remains the same. Therefore the secretory stimulants to the endothelium could not be the factor. The lymphagogue action of peptone remained absent when the liver lymphatics were ligated. This proved that the liver was the chief source of the increased flow in Heidenhain's experiments. Starling believes that it is the greater permeability of the capillaries rather than any increase in blood pressure which accounts for the increased flow of lymph.

Each of the factors presented in these theories may have some influence on absorption and exudation under certain conditions. Of these, no doubt, the last mentioned, the change in the vessel walls, is the most important. Changes in the vessel walls can no doubt increase their permeability. When such permeability is increased changes in pressure may be of influence. How much the activity of the endothelial cells may contribute is problematic.

III. **The Mechanism of Absorption.**—It is unlikely that the laws governing absorption from the surface of the peritoneum differ from those governing this process elsewhere in the abdomen. Most of the studies have been carried out upon the peritoneum because of the close analogy between this membrane and the artificial diaphragm of the physiologic laboratory. The following summary

is in agreement with Orlow. The process in general terms is as follows: When a hypertonic solution of NaCl is injected into the peritoneal cavity, fluid escapes from the blood into the peritoneal cavity as a result of a higher osmotic pressure. A part of the NaCl passes from the peritoneal cavity into the blood as a result of diffusion from the region of higher concentration to that of a lower. As a result of this process the hypertonic fluid becomes isotonic. If a hypotonic solution is employed it likewise becomes isotonic, but the processes are then reversed. Leathes and Starling demonstrated the plausibility of this hypothesis as follows: they placed 125 c.c. of 0.5 per cent NaCl solution in a parchment dialyzer and suspended it in a 0.5 per cent NaCl solution plus 10 per cent KNO_3. The next morning but 75 c.c. remained in the dialyzer, and the percentage of NaCl was the same, proving that not only fluid but salt, as well, had passed out of the membrane.

These factors, however, do not explain the absorption when once the solution has become isotonic. The researches of Starling and Cohnheim permit the following theory: The dividing membrane is impermeable to albumin, and water escapes out of the isotonic salt solution into the vessels. When this water is withdrawn, the solution in the abdomen becomes hypertonic again. Diffusion now of the NaCl into the vessels takes place until the fluid is again isotonic. The osmotic action of the albumin again takes place. These processes alternate until the fluid is absorbed.

Leathes and Starling used 60 to 100 c.c. of solution in the thoracic cavities of 4- to 8-kilo dogs and killed them after one-half to two hours and recovered the fluid. When the hypertonic solutions were used the amount of fluid recovered after two hours was greater than that used. When hypotonic solutions were used the amount recovered was less than that introduced. The rate of absorption was greater during the first half-hour than during the subsequent half-hour periods. Thus 39 per cent of the amount used had been absorbed at the end of the first half-hour, while at the end of two hours but 49 per cent had been absorbed. He believed that the lessened rate of absorption was due to the establishment of an osmotic equilibrium. When isotonic solutions are used the rate of absorption is the same during the first half-hour as during the subsequent half-hours up to two full hours.

This theory of absorption does not take into account the absorption of albuminous substances. Orlow calls attention to the fact that salt escapes into the peritoneal cavity when dilution of 0.3 per cent is reached while theoretically the change should begin at 0.5 per cent or 0.6 per cent. Orlow concludes, therefore, that the endothelial cells have an active part in the limitation of diffusion. According to Hamburger's studies this substance may affect the membrane mechanically. Leathes and Starling did not find that sodium fluoride lessened the rate of absorption except in so far as it made the solution hypertonic.

Starling and Cohnheim assumed that isotonic serums were absorbed by way of the lymphatics. However, Orlow found the lymph flow was not changed while serum was being absorbed, and Hamburger after tying the lymph vessels did not note diminution in the rate of absorption of the serum.

IV. **Avenues of Absorption.**—For many years a controversy waged as to whether fluid absorbed from the peritoneal cavity escaped into the lymphatics or into the blood vessels. It is now pretty definitely established that the greater portion, at least, escapes into the last named channels.

1. *Lymphatic Theory.*—That the lymphatics were the most active avenues of absorption was the most generally accepted theory immediately following the researches on "lymph production" just quoted.

The influence of these studies and an *a priori* reasoning on the hypothetic openings between the peritoneal surface and lymphatics can be traced through the writings of this period. Among the authors most prominent in assuming this view were Recklinghausen, Dybkowsky and Schweigger-Seidel. Of modern authors Cohnstein and Adler and Meltzer are the most prominent advocates. These investigators employed several methods. They tied the thoracic duct and introduced solutions of substances into the peritoneal cavity. They found that animals with open lymphatics responded more quickly to strychnine than when the lymphatics were tied. Adler and Meltzer based their opinion on the fact that when sodium ferrocyanide was injected into the peritoneal cavity this substance appeared later in the urine when the thoracic duct was tied, and that the substance appeared earlier in the lymph than in the urine.

In repeating Starling and Tubby's experiments Meltzer found that the lymph was colored in fourteen minutes while the urine was not colored for one and one-half hours. When 10 per cent potassium ferrocyanide was used it appeared in lymph in fourteen minutes and in urine in fifty-five minutes. They made the very significant remark that when the fluid was injected directly into the circulation it appeared in the lymph before it appeared in the urine. Therefore the experiments merely showed the time of secretion from the kidney and not necessarily a delay of entrance into the blood.

2. *Blood Vessel Theory.*—The early experiments of Magendie (quoted by Heidenhain) in which poisons were injected into tissues connected with the body only by blood vessels proved that absorption through these channels is possible. These studies were amplified by Asher and Hamburger who used potassium-iodide injections into a limb which had been severed from the body all save the large blood vessels and recovered the drug from the urine. Munk secured convulsions in the same experiment with the use of strychnine. These experiments may be easily repeated and bring assurance of the possibility of absorption through the blood vessels, a possibility Heidenhain once denied. That the blood vessels are the chief avenues of absorption was later maintained by Orlow, Starling and Tubby and Chittenden. Starling and Tubby had found, contrary to the conclusions of Adler and Meltzer, that dyes appeared in the urine before they did in the lymphatics. By inserting a cannula into the thoracic duct and one into the ureter they found that after injecting solutions of indigo carmine or methylene blue into the peritoneal cavity, the urine was colored in from five to twenty minutes while the lymph was not colored until one-half to four hours. They believe that the lymph may be colored by the coloring matter being first taken up by the blood stream and gaining access to the lymph vessels secondarily. After the paper by the last-named authors was published the work was repeated by Starling and Mendel and the original findings of Starling and Tubby were substantiated. The discrepancy according to the later authors was due to the fact that Adler and Meltzer removed the urine from the bladder instead of from the ureter. The amount of fluid collected from the thoracic duct is far below

the amount absorbed from the peritoneal cavity. Hamburger was the first to emphasize this very important point. Orlow obtained only 45 c.c. of lymph while 200 c.c. of fluid was being absorbed from the peritoneal cavity.

Dandy and Rowntree finally have decided the controversy in favor of the blood vessels. These investigators used phenolsulphonephthalein as an indicator. They used 0.6 mg. of this substance in 19 c.c. of 0.8 per cent salt solution injected into the peritoneal or pleural cavities of dogs. A fistula was made in the thoracic duct and the bladder was catheterized. The dye appeared in the urine in six minutes and in the lymph only after thirty-seven minutes. In an hour 56.4 per cent of the dye was regained from the urine, while less than 0.1 per cent was regained from the thoracic duct.

In another experiment the same solution was injected into the peritoneal cavity while 1 c.c. of a 4 per cent solution of indigo carmine and 19 c.c. of an 0.8 per cent NaCl solution was injected into the pleural cavity. The phthalein appeared in the urine in nine minutes and the indigo carmine in ten minutes. The phthalein appeared in the lymph in thirty-nine minutes and the indigo in forty-seven minutes. In an hour 50.2 per cent of the phthalein was recovered from the urine, and but a trace from the lymph. The indigo carmine imparted a deep blue to the urine and but a faint bluish color to the lymph.

The appearance of the phthalein in the blood was tested by examining the serum of the blood removed from the cavities at one-minute intervals after the injection of the dye as above noted. The phthalein appeared in the blood in four minutes and in the lymph in twenty-two minutes.

In view of the results obtained by these investigators, there can be no doubt that the chief avenue of absorption of isotonic fluids from the peritoneal cavity takes place by way of the blood vessels.

I can not suppress the suspicion that an attempt to measure the rate of absorption from the peritoneal cavity by collecting the outflow from the thoracic duct is bound to lead to erroneous conclusions. This duct should be regarded as primarily a collecting duct of the lacteals and as such measures the absorption from the nutritive material derived from the intestinal mucosa. The lym-

phatics of the peritoneum, as distinguished from the lacteals, empty primarily into the abdominal lymph glands and not into the thoracic duct. Not only my anatomic studies, but my clinical observations, have convinced me of the importance of this point. Malignant growths of the peritoneum metastasize in the lymph glands, as they do when there is a tuberculous lesion. Carcinoma of the thoracic duct is a rare lesion. Winkler could collect a bare dozen cases, and Schwedenberg could add but one to the list. Tuberculosis of the duct is found almost exclusively in miliary tuberculosis as shown by Longcope and these were secondary for the most part to retroperitoneal lymph gland tuberculosis.

V. **Rate of Absorption from the Peritoneal Cavity.**—The rate of absorption varies greatly according to the character and concentration of the fluid. There is always an initial rapid absorption in hypotonic solutions. Leathes and Starling found that 39 per cent of the amount injected is absorbed during the first half hour, while at the end of two hours but 49 per cent has been absorbed. This slowing is due to the establishment of an osmotic equilibrium. In isotonic solution the rate is the same for the various half-hour periods. Dybkowsky thought the pumping action of respiration accounted for the absorption.

In hypertonic solutions an actual exudate takes place into the pleural cavity as Leathes and Starling have shown.

Temperature within wide limits seems to have little influence. Roger introduced 120 to 250 c.c. of fluid at 0° C. (88 to 140 kilo weight) into the peritoneal cavity. No notable result was observed and the rate was not slowed.

Fleisher and Loeb found that absorption is increased in the nephrectomized animals and in those in which the renal vessels have been ligated. In general, those factors which raise the osmotic pressure of the blood hasten absorption. The use of adrenalin increases the rate of absorption by increasing the osmotic pressure.

My own studies on the rate of absorption were conducted by injecting a given amount of fluid into a series of animals of the same litter. After being injected they were killed at intervals, in most series, of thirty minutes. The percentage absorbed is decreased when the amount injected is excessive. Generally speak-

ing, if the amount of fluid injected does not exceed 10 per cent of the body weight, 30 per cent of the fluid injected will be absorbed in the first half-hour and at the end of two hours, less than 30 per cent will remain. Astonishing individual variations are encountered without there being a reason apparent. These occurrences are of interest because they lessen the value of experiments made in abnormal conditions.

VI. **Absorption of Solid Particles.**—The determination of the means by which the peritoneum disposes of solid particles is of more than theoretic interest. In many diseases insoluble particles remain and must be disposed of before complete restitution is possible.

Von Recklinghausen was the first to study the avenues of absorption of solid particles. He injected milk, cinnabar, lampblack and egg yolk into the peritoneal cavity of rabbits. He noted that the centrum tendineum became filled with those substances. He cut out a piece of diaphragm and covered it with a layer of milk and saw the globules collect about the stomata. Beck introduced blood into the peritoneal cavity and exposed the thoracic duct and saw blood escape from it. He also saw openings into the peritoneum which would admit several blood cells at a time. After two hours to four days he killed the animals, removed the diaphragm and stained with a solution of silver nitrate. He could note openings filled with particles. He could not find similar openings in the pleural cavity, but nevertheless, he found that particles escaped from the pleural cavity and were found in the kidneys. He injected alien blood into the peritoneal cavity and demonstrated its escape from the thoracic duct in two hours. MacCallum noted that when solid particles were introduced into the peritoneal cavity they were soon surrounded with phagocytes, which, when laden with these particles, escaped into the lymphatics through the roofs of the lacunæ and were swept to lymph glands. He found the granules still enter the lymphatics after the animal is killed. This continued even after the use of hot water and formalin. The granules outlined the cells nearly as completely as silver nitrate does. Southgate studied absorption of blood from the peritoneal cavity from another animal. All but one or two grams were absorbed after three hours. The lymphatics of the diaphragm were filled with

blood but the peritoneal lymphatics did not contain blood. He agrees with Ponfick in that the number of red cells in the blood was increased. When the animal is bled into its own abdomen the blood is readily absorbed without coagulation.

Auscher and Lapicque studied the fate of the pigment derived from the injection of blood into the peritoneal cavity. At first the greater number of granules were found in the spleen, but later they were more numerous in the liver.

Most authors describe the early localization of solid particles on the diaphragm. Launoy, however, found differently. He found the small particles held together by particles of lymph. The masses were found both above and below the great omentum. Many were located on the gastrosplenic and gastrohepatic omentum and the suspensory ligament of the liver. He found them particularly numerous about the testicle of guinea pigs. He thinks the so-called sweeping action of the omentum has nothing to do with the transplantation of these particles.

Buxton and Torrey present the most complete study yet published. According to these authors, shortly after the foreign bodies are introduced, the polynuclear leucocytes and hyaline cells disappear. This period of leucopenia lasts one to three or four hours, after which polynuclears reappear. In twenty-four hours the large mononuclears have englobed some of the particles, possibly previously taken up by polynuclear leucocytes. Further than this they do not trace the particle-laden cells. They found, by sections, that particles reach the spleen and liver as early as 15 to 30 minutes after the injections are made. This is sufficient proof that they were not conveyed there by cells.

By what avenue the particles reach these organs is not known. Maffucci found the position of the granules the same, whether the particles were injected into the peritoneum or the jugular vein. My own observation confirms the assumption that absorption takes place by way of the blood stream.

The conduct of cells native to the peritoneal fluid has been variously interpreted. Metchnikoff believed that the leucocytes are destroyed. Durham and Pierallini found that they are deposited on the great omentum. This may be qualified by saying that they are found wherever the particles are; for the most part on the

omentum, it is true. Their purpose there is, in my opinion, to form, together with the increased exudate produced by the irritation of the foreign body, the fibrin which encapsulates the foreign body. After this encapsulation is complete, cells again increase in numbers in the free fluid.

Dandy and Rowntree injected carmine and lampblack in watery suspension into the abdominal cavity by means of a trocar. The carmine was partly soluble and partly insoluble. The animals were allowed the freedom of the cage and after two or three hours a fistula was established in the thoracic duct. The lymph as obtained was faintly stained with carmine, but insoluble granules were absent. The lymphatics and glands were studied. They could detect no predilection for the anterior mediastinal lymph glands. Granules were rarely found in the liver and spleen. All lymph glands of the abdomen contained carmine, but granules were not found in these nor in the cisterna magna nor in the thoracic duct.

My own researches include the use of many substances and many species of animals. The most of the studies, however, were made with lampblack, carmine and nascent silver chloride. The animals employed were chiefly rabbits and guinea pigs, though many dogs and mice were used. A great variety also of the cold-blooded animals were employed. It may be added that there is no difference in principle. The cell-type active differs somewhat in the different species of animals.

The technic in all is much the same. Lampblack or carmine is rubbed up in sterile water, with or without the addition of gum arabic. The silver chloride was prepared by first injecting a solution of NaCl into the peritoneal cavity, and following this with less than the required $AgNO_3$ solution to produce a complete precipitation of the NaCl. The amount of these substances employed varied greatly. No notable differences were observed in the behavior of these substances.

The following outline presents the average findings: after these foreign bodies are introduced there is, first, a contraction of the vessels of the peritoneum, to be followed in a few minutes by a dilatation. With the dilatation there is an increased exudation into the peritoneal cavity. With this increase of exudation there is a sweeping of the particles toward the nooks and dependent por-

tions of the peritoneal cavity and about the great omentum. This may take place in any direction quite as pronouncedly toward the pelvis as towards the diaphragm. The great omentum and about the mesenteric root is apt to be the center of greatest accumulation. In fact, the material may be deposited anywhere. After observing many hundreds of abdomens in which injections of foreign particles have been made, I am disposed to say of their disposition what Treves said of the position of the appendix,—nothing is so constant as the inconstancy. The gut surface is usually relatively, but not absolutely, free. This is true whether the great omentum is retained or not.

With the localization of these particles there is a plastic exudate thrown about the granular mass which undergoes the usual steps toward the encapsulation, as is elsewhere described. This encapsulation required two or three hours, corresponding to the leucopenic stage of Buxton and Torrey already referred to. In this way a permanent resting place is assured. This process is altogether like soot granules in the lung or powder stains in the skin.

The process of exudation and fibrin encapsulation may be followed by employing Marchand's technic. This observer spread the mesentery of a small animal on a moist warm stage and then brought sterile foreign bodies on it and observed the changes.

In this manner one can note the increase in the exudate about the foreign body and the floating about of the foreign body in this fluid until anchored by the production of fibrin. The final stages of encapsulation can be followed only in stained sections.

The above described process represents the end fate of the great bulk of insoluble material introduced into the free peritoneal cavity.

Some of the finest particles are probably absorbed directly into the blood stream, but the amount is small.

The anterior sternal lymph gland receives some of the particles, but other glands of the abdomen likewise receive them. Pigments already in the lymph glands may cause confusion, as Muscatello has noted when lampblack is employed and basic tissue may become dyed when carmine is employed, thus being easily confused with carmine granules, as I have had occasion to note. The nascent silver chloride is more easily followed than either lampblack or carmine; and I have employed it largely in my studies. The use

of silver chloride, according to the method above mentioned, is artificial in that the normal state of the peritoneum is disturbed by the salt and silver nitrate solutions. These particles seem to pass directly into the blood stream. Most of the particles are found in the capillaries of the organ and but few in the lymph glands.

How these solid particles gain entrance to the lymph glands is a matter of speculation. In some cases the rapid appearance indicates that they may be swept into channels by pure mechanical processes; for the most part, however, the process is slower.

After the pigment masses are encapsulated in fibrin, or possibly while this process is going on, the number of cells in the peritoneal fluid is increased. The point of escape of these cells is at the area of the peritoneum irritated by the contact of the foreign body. The purpose of some of these cells is to form a new peritoneum for the capsules of the foreign bodies. While this encapsulation is taking place many of the particles are surrounded, "englobed" by the cells. The cells chiefly increased are the polynuclears and the large mononuclears. Early the polynuclears predominate, but later the mononuclears are numerous. It is a supposition that these cells laden with these foreign bodies gain entrance to the lymphatic circulation, and are carried to some permanent resting place in one of the lymph glands. It has seemed to me as though the surrounding of a small foreign body by the leucocytes was for the purpose of breaking it up, and that, failing in this the cells themselves are destroyed; while if the particles are soluble, as fibrin particles, the cell remains alive. This general impression is gained from *in vitro* studies. The large mononuclears could not be so studied because they could not be obtained for study.

The discrepancy observed in the rather voluminous literature is due apparently in the main to the different substances employed and the size of the particles. When very small particles are employed as filtered solutions of lampblack, no doubt many of the particles are swept into the circulation just as definitely as atoms in actual solution. Larger particles no doubt become surrounded by the protoplasmic mass of a cell or cells. What these cells do with these particles has not been satisfactorily demonstrated. It

is quite possible that they journey to a lymph gland and become "interned." Observation of lymph glands in pneumonoconiotic lungs is more convincing than any studies made on the peritoneum.

VII. **Factors which Hasten Absorption.**—Magendie (quoted by Heidenhain) proved that absorption from the pleural cavity was hastened if the volume of blood was reduced. He bled animals, and discovered that convulsions came on earlier when solutions of strychnine were placed in the pleural cavity.

Fleisher and Loeb are able to predict the influence of any procedure on absorption when its effect on the osmotic pressure of the blood is known. Nephrectomy and ligation of the renal vessels increase absorption, as predicted, since these procedures increase the osmotic pressure of the blood, and, true to the rule, increase the rate of absorption. I found that injecting hypertonic salt solution directly into the veins of animals acted in the same way.

Kellgren and Colombo studied the effect of massage on the rate of absorption. They found that massage always hastens absorption. Deep-stroking massage combined with kneading hastens absorption the most. Cohnstein found that massage increases the flow of lymph from the thoracic duct, presumably because it hastens absorption. Hanging the animal up by the hind legs likewise increases the flow. Heidenhain admits that there is an increased flow of lymph when the animal is so suspended, but denies that it is proved that the increased outflow of lymph is due to an increased absorption—a very pertinent observation indeed. Wegner found that an increase of the intraabdominal pressure hastened absorption so long as the increase in pressure is not great enough to retard the return flow of the blood. This fact has been generally overlooked. In intraperitoneal conditions in which intraabdominal pressure is increased where an increased absorption is not desired, caution should be exercised in reducing the increased intraabdominal pressure. For instance in peritonitis tympany probably is a conservative factor which acts by increasing intraabdominal tension. By incising the abdominal wall this pressure may be lessened and undesirable absorption increased. This point is difficult to prove for a degree of inflation simulating that in human diseases is difficult to imitate in animals. In rabbits the colon can be inflated

to a degree that markedly slows absorption. The use of hot injections and the scalding of the pleura did not lessen the rate of absorption, at least not until such a degree of heat was reached that albumin was made to coagulate. Schnitzler and Ewald found that increased peristalsis excited by ligation of the colon did not hasten absorption.

VIII. **Factors which Delay Absorption.**—Factors which delay absorption of fluids from the peritoneal cavity have been but little studied. Exudation into the peritoneal cavity seems hardly to have excited the interest of physiologists, except perhaps incidentally. This is unfortunate, since it is just this phase of the physiology of the peritoneum which is of the greatest value in the understanding of the diseases of the peritoneum.

Starling and Tubby showed that certain substances produced an exudate into the peritoneal cavity. When 50 c.c. of 10 per cent dextrose was injected, after one hour and fifty-five minutes, 96 c.c. of fluid was obtained and 50 c.c. of 10 per cent NaCl after three and one-half hours yielded 170 c.c. The greater amount of exudate after NaCl equals in osmotic pressure a 25 per cent sugar solution. This proves, they believe, that the escape of fluid is due to diffusion, and not to the activity of the blood vessels.

Schnitzler and Ewald used potassium iodide as an index. When dissolved in glycerine a slowing of the process resulted. When 10 c.c. of a 2 per cent aqueous solution was used, absorption was complete in from twenty to twenty-four hours. Alcoholic solutions gave variable results. When stronger solutions than the above are used, an actual exudation takes place. Schrader found that when 25 c.c. of glycerine was injected into a 16,000 gram dog 105 c.c. of fluid was obtained in forty-five minutes. This increase of fluid is a purely local reaction dependent on the physical properties of the glycerine. No exudate is produced when the glycerine is introduced subcutaneously.

Lessened peristalsis from the use of opium slowed absorption. It was slowed by injecting bacteria with the solution. Chronic peritonitis lessened the rate, as did extensive drying of the peritoneal surface.

Lazarus-Barlow found when using a segment of peritoneum that absorption of solutions of sodium chloride, sugar and urea

was slowed by the addition of albumin, a fact easy to confirm.

My own studies in this field aimed at (a) producing conditions in the peritoneal cavity inimical to absorption; and (b) producing conditions within the blood stream which hinder absorption from the peritoneal surface. In this field, fortunately, negative results are of interest and will be recorded.

Of the many substances used in these experiments compatible with the life of the animal, glycerine and formalin produced constant results. The former delays absorption for a time only. In solutions above 10 per cent an actual exudation may take place, and in weaker solutions delay in absorption uniformly occurs. In any strength, absorption finally takes place, and the entire amount of the fluid is absorbed. Formalin is more consistent. In a 2 per cent solution, absorption is prevented for from twelve hours to several days. This varies somewhat in like dosage in animals of the same species. Ordinarily a 50 per cent increase of the fluid takes place within a few hours.

The mode of action of each of these drugs is to produce an exudation of albumin on the peritoneal surface. It had previously been shown by Lazarus-Barlow that the addition of albumin to the fluids experimented with slows absorption. The addition of ascites or cyst fluid to saline solution causes marked slowing of absorption. Homologous tests with this material obviously can not be made. That ascitic fluid and cystic contents are ultimately capable of absorption is a matter of common clinical observation.

Formalin, in addition, exerts an influence on the peritoneal cells, acting, as it were, as a negative chemotactic force; that is to say, the normal number of fixed tissue cells is reduced. The amount of albumin exudation caused is considerable, sufficient to be readily demonstrable with ½ per cent solutions and with 5 per cent solutions the amount is sufficient to produce spontaneous coagulation when the fluid obtained from the peritoneal cavity is allowed to cool. Other drugs which produce a reactive exudation are considered in the section on chemical peritonitis.

None of the artificially induced blood conditions capable of slowing or preventing absorption from the peritoneal cavity, are compatible with the continued existence of the animal save artificially produced hydremia. Saline solution introduced into the

blood stream in any amount does not appreciably delay absorption. Elimination takes place so quickly that absorption goes on as before. A liter of 1:200 formalin solution introduced in a vein of a forty-pound dog delayed absorption several hours. Pronounced subcutaneous edema produced by injecting salt solution equal to one-fourth the body-weight of the animal slows the absorption for some hours. Thus 180 c.c. of normal salt solution injected subcutaneously in a guinea pig, in several regions of the body, will greatly delay, possibly for a time entirely prevent absorption. It is only by a combination of intravenous infusion and subcutaneous infiltration that the absorption can be slowed to actual inhibition. The amounts required to lessen the rate of absorption from the peritoneal cavity obviously could not be used in the human subject. If protoclysis is really of any benefit in peritonitis it is not by lessening the rate of absorption from the peritoneal cavity, and most certainly not by causing exudation into the peritoneal cavity as some credulous surgeons would have us believe.

IX. **Absorption of Blood from the Peritoneal Cavity.**—The elimination of accumulations of blood from the peritoneal cavity presents several points of interest. In it we have an example of rapid absorption of an isotonic fluid and the elimination of solid foreign bodies. In addition, these facts have an important clinical bearing.

The first to study this problem was Penzoldt and shortly after Poncet. This author demonstrated that defibrinated blood was completely absorbed from the peritoneum in from eight to thirteen days. Arloing (quoted by Poncet) transfused blood from an animal's carotid directly into its peritoneal cavity. In the dog they found clots along the vessels of the great omentum, but in rabbits absorption was complete. Toussaint (quoted by Poncet) injected 2 kilo 500 gm. of blood into the peritoneal cavity of an ass. He killed the animal after eight days and found the peritoneal cavity normal. Hayem showed that normal blood is absorbed as rapidly as defibrinated blood if the peritoneum is normal. These experiments were made without attempting to determine the exact amount of blood transfused. Lesage measured the amount of blood transfused from the carotid into the peritoneal cavity and determined the rapidity of absorption. He employed dogs weigh-

ing about 15 kilos and removed from 100 to 200 c.c. of blood from the carotid, and injected it into the peritoneal cavity. He calculated that this amount represented from one-eighth to one-fourth of the total amount of blood possessed by the animal. He noted that the formation of clot was very limited, seldom exceeding a few cubic centimeters but occasionally, the clot formed was more extensive. A large clot was noted in one animal in which there was a tumor. He noted that the clots were always attached to the great omentum, or that they lay near the pancreas. It is worthy of note, he says, that the formation of clot does not seem to retard absorption.

This author made accurate studies to determine the rate of absorption. After one hour the amount absorbed was not appreciable and after two hours, it was scarcely so. After three hours the amount absorbed was appreciable but not great. After this time has elapsed the blood is no longer spontaneously coagulable. After four hours there remain but a few cubic centimeters of blood and a small clot. In general, absorption begins at about four hours and is quite complete after forty-eight hours.

As might be expected, a knowledge of this rapid absorption led to a consideration of this avenue for the practice of therapeutic transfusion. Ponfick injected defibrinated blood and aside from a slight rise of temperature, no untoward phenomena developed. In order to determine the value of this method Bizzozero and Golgi found in twenty minutes after the injection there was an increase in the number of red cells. The maximum increase came after about two days. This increase in hemoglobin persisted as long as twenty-seven days. Obalinski found an increase in the red cells, beginning in an hour and continuing until absorption was complete. Nikolski found that when alien blood was used no such increase takes place, but instead, a hemoglobinuria is produced.

Southgate found absorption in progress in two hours and small amounts were completely absorbed in three hours. He noted that the lymph vessels of the pleural surface of the diaphragm were injected at the end of three hours. He found absorption less active if large amounts of blood were withdrawn. If alien blood was used, hemoglobinemia began in one hour and hemoglobinuria began in two hours.

The general opinion is that absorption takes place by way of the lymphatics and that it is taken over without material change in form or composition. One of the chief advocates of this view was Hayem as already noted. His opinion was based on an increased cell count after peritoneal absorption has begun. By injecting the blood of a dog, whose red cells have a diameter of 7 microns, into the peritoneal cavity of a kid, whose red cells have a diameter of 3.5 microns, he was able to demonstrate the red cells of the alien animal in the thoracic duct and blood vessels of the host.

This discussion is so much tinged with the prevailing views of stomata and lymphatic absorption that an extended discussion would be but a repetition of the account of absorption theories already presented. Lesage has varied the research by removing the thoracic duct at autopsy and examining its contents microscopically. He determined by this means that red cells may gain entrance to the thoracic duct in a normal condition, or be but slightly connoted. This author also tested the resistance of red corpuscles to lytic substances when obtained from the veins, carotids, and after a transfusion into the peritoneal cavity. He could detect no difference when obtained from a normal peritoneal cavity. When the peritoneum was diseased (tuberculosis) the red cells became more sensitive to lytic substances.

The early appearance of alien blood cells in the circulation as noted by Buxton and Torrey, indicates that these are directly absorbed by the blood stream. The absorption of noncoagulated blood seems to be similar to the absorption of isotonic fluids in general. The fibrin alone requires the slower action of phagolysis, though it has seemed to me that there may be fermentative changes, so to speak, which aid in liquefaction of the clot and thus hasten absorption.

X. **Site of Absorption.**—There is no more classical example of the far-reaching effect of reasoning on false premises than that concerned with the site of maximum absorption. Because of the technical simplicity of demonstrating the lymphatics of the diaphragm, as compared to other peritoneal surfaces, the impression early gained ground that this was the chief, if not the exclu-

sive, site in which they were to be found. The fictitious stomata were located here in superabundance.

Despite the fact that every physiologist knows that absorption takes place to a certain degree from any living tissue, and that surgeons observe every day that infective material located in any part of the peritoneal cavity is rapidly absorbed, the statement went unchallenged for many years that absorption from the peritoneal cavity takes place only from the surface of the diaphragm.

The anatomic side of this question receives appropriate attention in the chapter on histology, and it remains to be stated here only that solutions containing toxic drugs cause their characteristic physiologic action with equal rapidity, when area for area is considered. Dandy and Rowntree have recently contributed a careful study which showed that these fluids are absorbed with equal rapidity from all parts of the peritoneal cavity.

XI. **Effects of Inflammation on the Rate of Absorption.**—The effect of reactive processes on the rate of absorption is variable. The general opinion is that absorption is hastened. Whether or not this is true depends on the character and stage of the inflammatory process. In lower animals hypotonic irritants increase the rate of absorption, while hypertonic solutions delay it. Since a generalized inflammation can not be produced in the lower animals a study of the problem can not be made in conditions at all analogous to those found in the human subject.

In the human subject it is quite feasible to introduce a small amount of potassium iodide into the peritoneal cavity during the course of an operation, and so study the rate of absorption by the rate of its appearance in the urine. Only rough estimates are possible since there are no justifiable means of obtaining urine at sufficiently close intervals, save in those cases where a retention catheter is necessary. Methylene blue and phenolphthalein, being themselves antiseptics, can be introduced into the abdominal cavity without a therapeutic end in view. Employed in this way it may be categorically stated that in no condition of the peritoneum requiring surgical interference do these substances appear in the urine earlier than from the normal peritoneum. On the contrary, slowing always occurs depending on the degree of reaction. When there is an abundant fibrinous exudate, absorption is all but pre-

vented and this is also true in walled-off cavities. This method of studying this important question is worthy of the attention of surgeons who work in conjunction with competent chemists.

XII. **The Sensibility of the Peritoneum.**—A knowledge of the sensibility of the peritoneum, aside from its academic interest, is of value in the interpretation of diseased conditions which the sensation of pain enables us to make. Since abdominal operations are now being done under local anesthesia, this knowledge is of vital importance as a basis of technic. In this place only the theoretic phases will receive attention, the practical consideration being reserved for the chapter on peritonitis.

The question of the sensitiveness of the peritoneum has long been a question for study. Even the older literature contains interesting observations. Haller believed that the peritoneum had little or no sensation because the nerves apparently going to the peritoneum belong to the muscles adjacent to it. Bichat noted that organs supplied only by sympathetic nerves did not respond to the electrical, chemical, or mechanical stimuli. That no sensation was produced by injury he concluded because he saw dogs devouring their own intestines. Weber and Lennander believed that if pain is caused during an abdominal operation, abdominal or sacral nerves have been irritated. Bloch likewise believed that neither the visceral nor the parietal possess sensation. He did believe it has sensation when inflamed. Richet noted that the normal peritoneum is insensitive, or, at least, that the sensation is very obtuse, while it became "sensitive to painful excitations" when inflamed.

Kast and Meltzer found by animal experimentation that normal organs are sensitive which is much increased in inflammation. They assume the absence of sensation in the human when operated on under local anesthesia, and explain it on the ground of the constitutional anesthetic effect of cocaine. Ritter finds the organs of the dog to be sensitive. The lack of sensation found on opening the abdomen in humans under local anesthesia is due not to the general action of the cocaine, but to the action of the air upon the intestines, he believes. Wilms explains the absence of pain on touch and the presence of traction pains by the supposition that there are spinal nerves in the mesentery that end near the gut. Propping is of the same opinion.

The neural anatomy of the peritoneum will be discussed in the chapter on anatomy, and need not be anticipated here. It has been the failure to keep the elemental problem in neural anatomy in mind that has occasioned much of the discussion. When the functions of the peritoneum are considered the fact that the peritoneum does not respond to stimuli of touch, cold, etc., need occasion no more surprise than to find that it is devoid of auditory sensibility. The function of the peritoneal nerves can be none other than that of simple nutrition regulation. When the more deeply lying nerves are considered the cerebrospinal nerves are encountered in the abdominal wall and the sympathetic plexuses in the walls of the hollow viscera.

Broadly speaking, it may be stated that pain is caused whenever a sensory nerve is exposed to experiences of an irritating character to which it is not accustomed. To this it may be added that the mild stimuli which excite pain must be in the direction of an exaggeration of their physiologic function.

There can be no doubt that the parietal peritoneum is sensitive to all stimuli ordinarily affecting mixed nerves. For purposes of practice and experimentation the transversalis fascia and peritoneum must be considered as one structure. Since medullated fibers are found here in abundance this structure, as might be expected, is sensitive to manipulations. It is not sensitive to very light touch, that is to say, contact is not perceived as touch at all, but as pain, as soon as the contact reaches a certain degree of bruskness. Contact must exert its force as either pressure or traction. It is insensitive to pressure in proportion as the nerves are surrounded by soft tissues. An exposed membrane is not sensitive to weak pressure, as from a finger or glove, or to a piece of gauze. Traction causes pain when the normal tensile range of the surrounding tissue is exceeded. Before that point is reached a sensation is felt corresponding to the deep muscle sense of the extremities. This sensation corresponds to the sensation felt during violent peristalsis, movements of tumors in the abdomen, and, I presume, the fetal movements in pregnancy. The conclusions here recorded were gained by careful traction and palpation on the parietal peritoneum during the course of abdominal operations under quinine. The information has repeatedly been volunteered

by the patient, that light pressure is comparable to blunt pressure over muscles of the extremities. Most interesting was the statement of a woman of forty-six in whom I palpated the anterior abdominal region from the bladder to the umbilicus in the course of an operation for umbilical hernia. When asked to compare her sensations she remarked that it felt like fetal movements. Having had fourteen children she ought to know. Ramström believes it is the Pacinian bodies that transmit the sensation of movement of abdominal organs against the parietes.

The manipulations of the peritoneum are productive of much the same phenomena as those applied to an exposed nerve in an extremity. A common object of experimentation has been the ileoinguinal nerve exposed during the course of a hernia operation. One can pick it up, exert a considerable pressure upon it with the finger, or stick a needle into it without causing pain. It is only when considerable pressure, as with a forceps, is applied or traction made that pain is elicited. Pressure upon the nerve, if gently made, is not perceived at all. Muscle sense, apparently, can be elicited only when the muscle-end organs are involved, so that, strictly speaking, it is likely not a function of peritoneal nerves, but of those muscle nerves derived from the plexus from which the peritoneum receives its nerves.

The parietal peritoneum is not sensitive to the prick of a fine needle. Because of the relatively large meshes of the plexus the needle may penetrate without striking a nerve. In suturing the peritoneum it is the element of traction that causes pain. Only when suturing is very gently done, and there is no muscle tension, can pain be avoided.

The visceral peritoneum contains no medullated fibers. The attitude of some writers who assume that sympathetic nerves can not transmit painful stimuli, and agree, therefore, that medullated fibers must reach the viscera, is wholly without reason. Careful histologic examination has failed to demonstrate medullated fibers in the sympathetic bundles, and, until positive evidence is presented of their existence, it is fair to assume that they are not present. Since pain in the viscera is perceived it follows that, so far as our present knowledge goes, sympathetic fibers do transmit painful stimuli.

As noted in the chapter on anatomy, the gut tract is possessed of two plexuses. So far as the physiology of these nerves is concerned it may be said that they preside over the motor and secretory functions of the gut tract. Whether the reflexes that call forth movements of the intestine always involve the sensory neurons in the cerebrospinal ganglia, as maintained by Langley, or, as now seems more probable, it may take place through local reflex areas as indicated by the work of Bayliss and Starling and Cannon and Auer, is not known. The last-named authors showed that normal mobility of the digestive tube may be carried out for a considerable length of time when the splanchnic nerves are severed from the spinal ganglia. The correctness of the latter authors' views is made more probable by the recent work of Kuntz, who makes it appear that the local ganglia control the local reflex areas. These observations are important in the study of the origin of pain.

Many authors, notably Lennander, maintain that pain is caused only when traction is made on the mesentery and transmitted hence to the spinal ganglia. One author goes even to the extreme of assuming that in spontaneous gaseous cramps the pain results from traction of the distended loops on the mesentery. Experimental evidence, however, shows that pain may be caused when there is no traction made on the mesentery.

My studies on this point have been carried out particularly on the gall bladder. Repeated histologic and anatomic studies failed to show medullated fibers going to this viscus. During the course of gall bladder operations it is easy to demonstrate that pain is caused by distention of it, as, for example, with a fluid or forceps. This pain must be transmitted through the sympathetics.

Distention of the colon with air through a colostomy opening and of the small intestine through an enterotomy wound cause "cramps" transmitted mostly to the epigastrium, as may be easily demonstrated.

That the nerves of this region should be sensitive to overdistention, both in setting contractions into action and as pain, seems quite in harmony with the function that might be attributed to them.

As to the sensitiveness of the gut wall to direct mechanical

trauma, practical experience in abdominal operations furnishes an abundant array of data. A loop of gut may be pinched with moderate firmness without causing pain. Crushing causes but slight pain in the normal or edematized gut. Traction on the gut itself, independent of traction on the mesentery, causes pain. The base of the appendix, for instance, may usually be clamped without causing pain in an interval operation. Ligation of the mesentery causes little or no pain unless traction is made at the same time. The slight degree of compression necessary to set clamps for enteroanastomosis causes no pain. Cutting the gut may or may not cause pain, and, when present, the pain is usually but slight. Dilating the opening made by incision is attended by acute pain. In other words, the sympathetic nerves may be constricted, even crushed, without causing pain, but traction causes pain.

Suturing the gut is not attended by pain. This is easily understood. The plexus here is relatively large, and the chance of the needle point missing a nerve is vastly greater than the chance of hitting it. The compression of the suture is not great enough to cause pain in a normal gut wall. In interpreting pain sensations expressed by patients being operated on under local anesthesia, care must be exercised. Patients improperly prepared may start, or even cry out, when the finger touches the normal skin. Statements made by patients when in such a mental state are, of course, valueless. The operator must be able to judge whether his patient is of sufficient intelligence and is in the proper mental state to make the proper interpretations.

When the peritoneum is in a state of inflammatory reaction a different problem presents itself. The sensitiveness of the parietal peritoneum is much heightened, but not nearly so markedly as the sensitiveness of the visceral. When inflamed the gut is painful to contact, irrespective of traction on the mesentery. Inflammatory adhesions when separated cause acute pain. The clamping of inflamed guts causes pain. Packing off an inflamed area causes acute pain.

These observations may be summarized by saying that in the normal gut pain is caused only when traction much beyond the normal is made. In that state a registration of overdistention meets all the requirements of physiologic function. When inflamed, rest

is required, therefore pain is caused. No more pain is caused than is required to safeguard the abdominal organs.

I have endeavored to trace the changes in the tissues antecedent to the advent of the inflammatory sensibility. In general, it may be said that the same laws govern here that govern the change in sensibility in the somatic system relative to reactive processes. Simple hyperemia, either active or passive, does not increase sensibility in the absence of exudation. This is true whether the vascular dilatation is due to an irritant or to reflex causes. With the beginning of exudation sensation is heightened. This applies to both cellular and serous exudates. So far as my experience permits me to judge, the stage of inflammation in which the connective tissue is surrounded by an inflammatory exudate, but retains its specific tinctorial reaction, is characterized by sensibility. This corresponds to the height of tissue reaction against injury. This stage, once past sensitiveness, lessens. The connective tissue lessens its affinity for acid dyes, and approaches a fibrin state. In this stage it may be relatively free from sensation. The same may be true of tissue in a state of soggy edema when infected with organisms of intense virulence. As tissues become normal, sensitiveness returns and may equal that of the acute stage of inflammation. This may be noted in separating adhesions about the gall bladder.

These data were obtained by observing the state of reaction of the peritoneum at the time of operation, and comparing the data thus obtained with the microscopic picture of tissue removed. Data thus obtained need to be verified by other observers, for conclusions formed on a long series of observations if variable, must be subject to the errors of the personal equation of both the patients and the observer.

The Great Omentum.—Nerves have not been demonstrated with certainty in the great omentum. The difficulty of differentiating elastic fibers from sympathetic nerve trunks makes positive evidence difficult to obtain. This organ is relatively insensitive to manipulations. Ligation and resection may be freely done without eliciting the attention of the patient. In the inflamed state the omentum is nearly always adherent to some other peritoneal surface, so that pain elicited during manipulation may be the result

of irritation of the visceral or parietal nerves of the organ to which it is attached. The sensitiveness, even in a state of reaction, must be slight, for, in strangulated omental or umbilical or inguinal hernias, if the parietal peritoneal nerves are properly anesthetized, pain is not caused by the manipulation of the omental mass.

XIII. **Physiology of the Great Omentum.**—Hippocrates speculated on the function of the great omentum, and we are emulating his achievement. Since his time every possible hypothesis has been advanced, varying from the opinion of Erisistratus, who believed that it had no function, to some of our modern fantastic writers who speak of the "intelligence" of the omentum, implying thereby that a treatise on the psychology of the omentum is desirable, and it is probably only a matter of time until some one will present us with one. Such writings, while not enlightening us on the use of this interesting organ, do give us a line on the psychology of the writer. The omentum is not the only organ of which so much might be said.

Broman, in a monumental work, has taken the trouble to record many of the theories of the function of the omentum, dividing them into fifteen groups. A few of these groups may be quoted. The theory advanced by Fabricus ab Aquapendente appeals to me particularly. His idea was that air gained entrance to it which was used by ventriloquists. It seems to have this function in some of our political spell-binders; this is, no doubt, where the designation "windjammer," sometimes applied to these persons, originated. Laurembergius believed it condensed into fat oil vapors given off from the intestine, while de Zerbis believed it gave off fat to the intestine, which liquefied the feces. This is interesting as anticipating the use of paraffin oil, an idea which advertisement writers have overlooked. The last-named author believed it also protected the intestine from mechanical injury. It is probably this fact that suggested to Boerhave that its function is to work over the raw material of the gall; but into what, however, he wisely suppressed. A large number of writers, among them Aristotle and Galen, regarded the omentum as a protector against cold. Bauhin and Glisson regard it as a fat reservoir. The only reason for according the omentum this function is that it contained fat.

It can have no greater function than a deposit of fat elsewhere. That it is a fortification against anything is unlikely. An undue deposit here, as elsewhere, is unquestionably deleterious. Certainly, on the function *per se* the fat exerts a deleterious influence. Glisson and Petit believed it facilitated peristalsis; others regarded it as a suspensory ligament for the colon and as a tractor to the stomach to hold it down, and thus facilitate diaphragmatic respiration. The small flap formerly attached to the bosom of shirts to hold them down evidently originated from this notion of the function of the omentum.

From the foregoing it becomes obvious that it is easier for us to determine what the omentum does not do than to demonstrate what its real function is. Albrecht compared it to a large leucocyte, and Morison designated it the "policeman of the abdomen." Both views are fantastic, but probably point the way. Koch speaks of it as a lymph gland, and Weidenreich regards it as a flat lymph gland. Be this as it may, the only obvious function of the omentum is as a protective and reparative organ. This phase will be discussed in the chapter on inflammation.

It has been objected that no organ is provided to perform a pathologic function only (Heusner). It has not been demonstrated that leucocytes have any other function than that of reparative agents. This is not a valid objection, however, since repair is as much a function of the adult body as growth is of the fetus, and the two processes are probably closely related, the first, following a common law provided by inheritance, proceeded to a definite end, while the latter is set in motion by agencies which require its action for but a limited extent over a limited period of time. This view finds support in the simple fact that the above is what the omentum actually does.

That the omentum is not a vital physiologic organ is self-evident, for it is sometimes congenitally absent and it may be removed without discommoding the animal appreciably.

From the foregoing it is evident that the discussion of the function of the peritoneum falls largely under the head of pathology. A number of facts relative to the physiology of the omentum have been determined, which may be discussed under a number of heads, notably as to its capacity for absorption and to its capacity for

movement. Its capacity for movement and its function as a protective organ can best be considered under the subject of inflammation. Aside from the function it actually performs there are a number of functions it is alleged to perform, which may be given brief consideration. Most interesting of these is the blood-regulating and mechanical function.

The Omentum as a Regulator of Blood Pressure.—Rivinus and Ziegerus believed the omentum acted as a blood-regulator. Witzel believes so still. Broman regards it as possible that this may be an accessory function, but he points out the unexplained variability of its size in the various orders of mammalia. Gundermann distended the stomachs of etherized animals with air, and noted a slight dilatation of the upper omental veins in response to a dilatation of the gastric veins. He concluded, therefore, that there is a physiologic relationship.

These observations were made under very unphysiologic conditions. An animal asleep under morphine and ether with its stomach distended with air, does not present physiologic conditions. My studies were carried out under two conditions; first, the observation of the omental vessels in many animals (including humans) under varying conditions. In animals the conditions in the functionating and resting stomach may be observed in like animals. All sorts of adventitious conditions can be produced and conditions observed. The sum total of these observations fails to show any relationship between the omental vessels that would tend to show any compensating action. The other method of study was by placing a glass window in the abdominal wall and viewing the omentum direct. This method imposes an artificial link in the chain, but in so far as the method is trustworthy, it presents no evidence that can be construed as favoring a regulating action on the part of the omental vessels. In fact, the only factor that produced a dilatation was one that excited local reaction or hindered a return flow.

Much talk has been indulged in relative to the reservoir action of the omental action in shock. Actual observations calculated to form a basis for these conclusions are wanting.

The relation of resection of the omentum to gastric hemorrhages likewise throws but little light on the alleged blood-regulating

function. Von Eiselsberg has had seven such cases of gastric hemorrhage, in six of which some sort of operation was done on the omentum. He ascribes the cause of the hemorrhage to emboli from the region of operation. Friedrich also studied the problem of icterus as it relates to operations on the omentum. He noted the frequent occurrence of liver necrosis and ulcers of the stomach. He believes that portal thromboses resulting from the ligation of a vessel account for the phenomenon. In all the cases reported in these two papers the patients were males between the ages of 43 and 60. My observations are limited to two cases, both females, one aged 58 with a myoma of the uterus to which the omentum was attached, the other aged 32 was operated on for an abdominal pregnancy of the eighth month.

The observations above noted, namely, that the disease tends to affect adults, recalls the studies of Hochstetter who found valves in fetuses, and none in adults. Animals, furthermore, in which the valves were found, did not develop hemorrhages after the removal of the omentum, while those which did not possess valves, did develop hemorrhage.

Interesting as all these observations are, they bear little, or not at all, on the problem under discussion. Operations on the omentum are followed only at rare instances by gastric hemorrhage, and in these there is no evidence that changes in the statics of the circulation had anything to do with the hemorrhage.

Dilatation of the omental vessels in cirrhosis of the liver appears to be purely mechanical. The attachment of the omentum to uterine myomas mentioned by Gundermann certainly is a manifestation of its response to injury, and, in no sense, evidence of a blood-regulating function.

Absorption by the Omentum.—That absorption may take place through the omentum may easily be demonstrated by suspending the organ in a solution of strychnine. Convulsions will take place. It is difficult to be certain that none of the fluid has gained access by way of the peritoneum, however. The amount of fluid absorbed varies greatly in animals of the same size and species under like conditions. The fact that there is so great variation makes it probable that the experiments lack in exactness. The absorption by the omentum is slower than that of other peritoneal surfaces

of like extent. Rubin tested the rate of absorption in cats, then removed the great omentum, and after eighteen days retested the rate of absorption. He found it was delayed. The delay is very slight, and in animals in which a long interval between the resection and the absorption experiments has elapsed, it is not at all notable. Wilkie found no appreciable slowing of absorption from the peritoneal cavity in animals from which the great omentum had been removed three weeks before. He places the ratio as 3 to 2. Dunsmoor placed the rate of absorption through the omentum at 0.5 per cent of the body-weight per hour.

The most striking function of the omentum observed in physiologic experiments is the manner in which it deals with solid particles. When insoluble substances are introduced into the peritoneal cavity they are apt to be found in contact with the great omentum. This observation led Koch to conclude that the great omentum is the most important factor in absorption. Large substances, such as discs of corn pith, are usually fully surrounded by the omentum. It seems most likely that the adhesions of foreign bodies are an evidence of facile response to injury, rather than of capacity of absorption.

Mechanical Physiology.—The idea of mechanical function arises from the fact that the greater colic omentum serves as a suspensory ligament to the colon. Hansen (cited by Broman) advanced the idea that it exerted traction caudalward on the stomach. Recently Heusner from the study of the lower animals, again emphasizes the importance of the mechanical factor. Anchored by the weight of the intestines it serves to hold down the stomach and colon, so that less movement is imparted to these organs when the abdominal muscles are made tense by strenuous exertions.

Heger advanced the idea that the great omentum swept the intestines. He placed glass beads in the peritoneal cavity and they became attached to the great omentum. The movements of the beads could be observed by the x-ray. He believed that they were thus moved toward the diaphragm. It is quite true that when certain foreign bodies are placed in the abdomen, the surface of the intestine is freer from particles than is the omentum of animals from which the omentum has previously been removed. It is possible that this is due as much to the fact that the particles have be-

come adherent to the omentum as that the omentum has mechanically removed them. That the omentum moves the particles toward the diaphragm can, at least, be only partly true, for quite as many particles move toward the pelvis and other recesses as toward the diaphragm.

Movements of the Omentum.—That the normal omentum can not have spontaneous movements should require no argument. Its movements in pathologic conditions, it is interesting to note, have been ascribed to the action of muscle fibers in the blood vessels. Excited peristalsis does not move it, nor can it be made to move by artificial stimulation, as Rubin has shown. A discussion of its migration may, therefore, be more properly taken up in the section on pathology.

Bibliography

Adams: Peritoneal Adhesions, Lancet, London, 1913, i, 663.

Adler and Meltzer: Experimental Contribution to the Study of the Path by which Fluids are Carried from the Peritoneal Cavity into the Circulation, Jour. Exper. Med., 1896, i, 482.

Asher: Ein Beitrag zur Resorption durch die Blutgefässe, Ztschr. f. Biol., 1892-3, n.F., xi, 247.

Auer: The Effect of Severing the Vagi or the Splanchnic or Both upon the Gastric Motility of Rabbits, Am. Jour. Physiol., 1909-10, xxv, 334.

Auscher and Lapicque: Localisation de le rubigine produite par injection de sang dans de peritoine, Compt. rend. Soc. de biol., Paris, 1898, 10 s., v, 185.

Bayliss and Starling: Movements and Innervation of the Small Intestines, Jour. Physiol., 1889, xxiv, 99; ibid., 1900-01, xxiv, 125.

Beck: Ueber die Aufsaugung ein vertheilter Körper aus der serösen Höhlen, Wien. klin. Wchnschr., 1893, vi, 823-824.

Bichat: Traite des membranes in général et des diverses membranes in particular, Gabon, Paris, 1827.

Bizzozero and Golgi: Della transfusione del sangue nel peritoneo e della sau influenza sulla ricchezza globulare del sangue circolante, Arch. per le sc. med., 1880, iv, 67.

Bloch: Mécanisme de la quérison de la péritonite tuberculeuse par la laparotomie, Cong. internat. d. méd. C. d., Par., 1910, sect. de therap.; p. 485.

Broman: Die Entwicklungsgeschichte der Bursa Omentalis, Bergmann, Wiesbaden, 1904, p. 571.

Brücke: Vorlesungen über Physiologie, Braumüller, Wien, 1874, i, 190.

Buxton and Torrey: Absorption from the Peritoneal Cavity, Jour. Med. Research, 1906, xv, 1.

Chittenden (*et al*): A Chemicophysiological Study of Certain Deviations of the Proteids, Am. Jour. Physiol., 1899, ii, 142.

Cohnheim, J.: Lectures on General Pathology, London, New Syndenham Soc., 1889.

Cohnstein: Zur Lehre von der Transsudation, Virchows Arch. f. path. Anat., 1894, cxxxv, 514.

Ueber Resorption aus der Peritonealhöhle, Zentralbl. f. Physiol., 1895, ix, 401.

Cannon: The Motor Activities of the Stomach and Small Intestines after Splanchnic and Vagus Section, Am. Jour. Physiol., 1906, xvii, 429.

Dandy and Rowntree: Peritoneale und pleurale Resorption in ihren Beziehungen zu der Lagerungsbehandlung, Beitr. z. klin. Chir., 1913, lxxxvii, 539.

Dunsmoor: The Versatile Omentum, Tr. West. Surg Assn., 1908, xviii, 323.

Durham: The Mechanism of Reaction to Peritoneal Infection, Jour. Path. and Bacteriol., 1896-1897, iv, 338.

Dybkowsky: Über Aufsaugung und Absonderung der Pleurawand, Arb. a. d. physiol. Anst. zu Leipz., (1866), 1867, i, 40.

v. Eiselsburg: Magen und Duodenal-blutungen nach Operationen, Arch. f. klin. Chir., 1899, lix, 837.

Emminghaus: Ueber die Abhängigkeit der Lymphabsonderung vom Blutstrom; Arb. a. d. physiol. Anst. zu Leipz. (1873), 1874, viii, 51.

Fleisher and Loeb: Absorption from the Peritoneal Cavity, Proc. Path. Soc., Philadelphia, 1910, n.s., xiii, 56.

Friedrich: Zur chirurgischen Pathologie von Netz und Misenterium, Arch. f. klin. Chir., 1900, lxi, 998.

Gundermann: Uber die Bedeutung des Netzes in physiologischer und pathologischer Beziehung, Beitr. z. klin. Chir., 1913, lxxxiv, 587.

Haller: Element physiologiae corporis humani, Lausanne u. Bern, 1757-1766.

Hamburger: Über die Regelung der osmotischen Spannkraft von Flüssigkeiten in Bauch- und Perikardialhöhle; ein Beitrag zur Kenntnis der Resorption, Arch. f. Physiol., 1895, 281.

Hamburger: Über die Regelung der Blutbestandtheils bei experimenteller hydrämischer Plethora, Hydrämie und Anhydrämie, Ztschr. f. Biol., 1890, n.F., ix, 259.

Ueber Resorption aus der Peritonealhöhle, Centralbl. f. Physiol., 1895, ix, 484.

Hayem: De la transfusion péritonéale, Compt. rend Acad. d. sc., 1884, xcviii, 749.

Heger: Le balavage de la cavité péritonéale par l'épiplon, Arch. internat. de physiol., 1904, 1, 26.

Heidenhain: Versuche und Fragen zur Lehre von der Lymphbildung, Arch. f. d. ges. Physiol., 1891, xlix, 209.

Bemerkungen und Versuche, betreffs der Resorption in der Bauchhöhle, Arch. f. d. ges. Physiol., 1895, lxii, 320.

Hertzler: A Possible Danger in the Therapeutic Use of Osmic Acid, Med. Rec., 1906, lxx, 380.

The Anatomy and Physiology of the Peritoneum and Their Relation to Peritonitis, Tr. West. Surg. Assn., 1903, xiii, 311.

Heusner: Die physiologischen Bedeutung des Grossen Netzes, München. med. Wchnschr., 1905, lii, 1130.

Hochstetter: Die normale Vorkommen von Klappen in den Magenverzweigungen der Pfortader beim Menschen und einigen Säugethieren, Arch. f. Anat. u. Entwcklngsgesch., Leipz., 1887.

Kast and Meltzer: Die Sensibilität der Abdominalorgane und die Beeinflussung derselben durch Injektionen von Cocain, Berl. klin. Wchnschr., 1907, xliv, 600.

Kellgren and Colombo: Du role que jouent lymphatiques et les veins dans de la Absorption des Exudations, Compt. rend. Soc. de biol., Paris, 1895, 10 s, ii, 463.

Koch: Über des Verhalten des Grossen Netzes bei der peritonealen und intestinalen Infection, Med. Klin., 1911, vii, 1912.

Kuntz: On the Innervation of the Digestive Tube, Jour. Comp. Neurol., 1913, xxiii, 173.

LANGLEY: On Reflex Action from Sympathetic Ganglia, Jour. Physiol., 1894, xvi, 410.

LAUNOY: Sur la localization des particules fines injectés dans la péritoine de cobyaye male, Compt. rend. Soc. de. biol., Par., 1908, lxv, 605.

LAZARUS-BARLOW: Observations upon the Initial Rates of Osmosis of Certain Substances in Water and in Fluids Containing Albumen, Jour. Physiol., 1895-6, xix, 140.

LEATHES AND STARLING: On the Absorption of Salt Solutions from the Pleural Cavities, Jour. Physiol., 1895-6, xviii, 106.

LENNANDER: Beobachtungen über die Sensibilität in der Bauchhöhle, Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1902, x, 38.

LESAGE: Recherches expérimentales sur la resorption du sang par le peritoine, These, Paris, 1902.

LONGCOPE: Tuberculosis of the Thoracic Duct and Acute Miliary Tuberculosis, Am. Med., 1906, xi, 16.

LUDWIG AND SCHWEIGGER-SEIDEL: Ueber das Centrum tendineum des Zwerchfells, Arb. a. d. physiol. Anst. zu Leipz., (1866), 1867, i, 174.

MACCALLUM: On the Relation of the Lymphatics to the Peritoneal Cavity in the Diaphragm and the Mechanism of Absorption of Granular Materials from the Peritoneum, Anat. Anz., Jena, 1903, xxiii, 157.

MAFFUCCI: Esperimenti sull assorbimento del peritoneo, Gior. internaz. d. sc. med., 1882, n.s., iv, 657.

MARCHAND: Ueber die Veränderung der Peritoneal Endothelien bie der Einheilung Kleiner Fremdkörper, Sitzungsb. d. Gesellsch. z. Marburg, 1897, No. 3, p. 29.

MELTZER: On the Paths of Absorption from the Peritoneal Cavity, Jour. Physiol., London, 1897-8, xxii, 198.

MENDEL: On the Absorption from the Peritoneal Cavity, Am. Jour. Physiol., 1898-9, ii, 342.

METCHNIKOFF: L'Immunite, p. 86.

MORISON: Remarks on Some Functions of the Omentum, Brit. Med. Jour., 1906, i, 76.

MUNK: Zur Kenntnis der interstiellen Resorption wasserlöslicher Substanzen, Dubois' Arch., 1895, p. 387.

MUSCATELLO: Ueber den Bau und des Aufsaugungsvermögen des Peritonäum, Virchows, Arch. f. path. Anat., 1895, cxlii, 327.

NASSE: Untersuchungen über die Einflüsse, welche die Lymphbildung beherrschen, Marburg, Elwert, 1871.

NIKOLSKI: Ueber den Einfluss von Blutinfusion in die Bauchhöhle auf die Menge der Blutkörperchen und des Hämoglobulin im cirkulirenden Blute, Centralbl. f. Chir., 1880, vii, 315.

OBALINSKI: Doswiadczalny przyczynek do przetaczania krwi do jamy otrzewnowéj. (Resorption of ecchymosis.) Prezgl. lek., Krakow, 1880, xix, 117, 133.

ORLOW: Einige Versuche über die Resorption in der Bauchhöhle, Arch. f. d. ges. Physiol., 1894, lix, 170.

PENZOLDT: Ueber das Verhalten von Blutgefässen in serösen Höhlen, Deutsches Arch. f. klin. Med., 1876, xviii, 542.

PIERALLINI: Sur la phagolyse dans la cavité péritonéale, Ann. de l'Inst. Pasteur, 1897, xi, 308.

PONCET: Le l'hématocele des péri-utérine, Thèse, Paris, Germer-Bailliere, 1878, and Dictionaire des Sciences med., 1886, xii, 736.

PONFICK: Experimenteller Beiträge zur Lehre von der Transfusion, Virchows Arch. f. path. Anat., 1875, lxii, 237.

Ueber ein einfaches Verfahren der Transfusion beim Menschen, Jahresb. d. schles. Gesellsch. f. vaterl. Kult., 1880, lvii, 60.

PROPPING: Zur Frage der Sensibilität der Bauchhöhle, Beitr. z. klin. Chir., 1909, lxiii, 690.

RAMSTROM: Anatomische und Experimentelle Untersuchungen Über die Lamellösen Nervenendkörperchen im Peritoneum parietale des Menschen, Anat. Hefte, 1908, xxxvi, 309.

V. RECKLINGHAUSEN: Lymphgefässystem in Strickers Handbuch der Lehre von den Geweben, Leipz., 1871, 214.

Zur Fettresorption, Virchows Arch. f. path. Anat., 1863, xxvi, 72.

RICHET: Dictionnaire de Physiol., Paris, 1900, lv, p. 186.

RITTER: Experimentelle Untersuchungen über die Sensibilität in der Bauchhöhle, Centralbl. f. Chir., 1909, vi, Beitr. 57, Verhandl. d. deutsch. Gesellsch. f. Chir., 1909, xxxviii, 514.

ROGER: Note sur les effets des injections d'eau glacée dans les veines, ler paritoine, Compt. rend. Soc. de biol., Paris, 1897, 10 S., iv, 695-697.

ROGOWICZ: Beiträge zur Kenntnis der Lymphbildung, Pflügers Arch., 1885, xxxvi, 152.

RUBIN: The Function of the Great Omentum, Surg., Gynec., and Obst., 1911, xii, 117.

SABIN: The Origin and Development of the Lymphatic System, Balt., Johns Hopkins Press, 1913.

SCHNITZLER AND EWALD: Ueber experimentelle Beeinflussung der peritonealen Resorption, Wien. klin. Rundschau, 1895, ix, 273.

SCHRADER: Experimentelle Beiträge zur Kenntnis des Transudationsvonganges am Bauchfell, Deutsch. Ztschr. f. Chir., 1903, lxx, 421.

SCHWEIGGER-SEIDEL AND DOGIEL: Ueber die Peritonealhöhle bei Fröschen und ihren zusammenhang mit den Lymphgefässystem, Arb. a. d. physiol. Anst. zu. Leipz. (1866), 1867, p. 68.

SCHWEDENBERG: Ueber die Carcinose des Ductus Thoracicus, Diss. Berlin, 1905.

SOTNITSCHEWSKY: Ueber Staungsödem, Virchows Arch. f. path. Anat., 1879, lxxvii, 85.

SOUTHGATE: Ueber Blutresorption aus der Peritonealhöhle, Centralbl. f. Physiol., 1894, viii, 449.

STARLING: On the Mode of Action of Lymphagogues, Jour. Physiol. 1894-1895, xxiv, 30.

On Absorption from the Peritoneal Cavity, Proc. Physiol. Soc., Lond., 1897-8, xxiv.

STARLING AND TUBBY: On the Paths of Absorption from the Peritoneal Cavity, Jour. Physiol., Lond., 1897, xxii, 198-205.

On Absorption from and Secretion into the Serous Cavities, Jour. Physiol., 1894, xvi, 140.

TOMSA: Beiträge zur Lymphbildung, Sitzungsb. d. kais Akad. d. Wiss., Wien, 1893, xlvi, 185.

WEBER: Der Tastsinn und das Gemeningefühl, Wagner's Wörterbuch d. Physiol., Braunschweig, 1850, iii, 2 Abt. 481.

WEGNER: Chirurgische Bemerkungen über die Peritonealhöhle mit besonderer Berücksichtigung der Ovariotomie, Arch. f. klin. Chir., 1877, xx, 51.

WEIDENREICH. Die Leucocyten und verwandete Zellformen, Bergmann, Wiesbaden, 1911.

WILKIE: Some Functions and Surgical Uses of the Omentum, Brit. Med. Jour., 1911, ii, 1103.

WILMS: Ueber die Sensibilität und Schmerzempfindung der Bauchorgane, Deutsch. Ztschr. f. Chir., 1909, c. 372.

WINKLER: Ueber die Beteiligung des Lymphgefässystems an der Verschleppung bösartiger Geschwulste, Virchows Arch. f. path. Anat., Suppl. cli, p. 195.

WITZEL: Die Schutzarbeit im Bauchraume und die functionelle Behandlung Laparotomierter, München. med. Wchnschr., 1909, lvi, 269.

CHAPTER II

HISTOLOGY OF THE PERITONEUM

Scope.—The structures which are regarded as constituting the peritoneum have been the subject of a wide range of interpretation. In the discussion of the peritoneum's minute anatomy, histologists have been prone to include only the cells and the basement membrane, while gross anatomists and surgeons have been disposed to adopt a wider interpretation, and have included, not only the structures above mentioned, but also those lying between it and the underlying parenchymatous organs. The too great limitations of the concept of the former are at once apparent, for the lymph vessels lying beneath the basement membrane have, very commonly, been discussed in their anatomic and physiologic relations to the functions of the peritoneum. That the peritoneum can not be considered apart from its vessels will become apparent at once when the pathology of the peritoneum is considered. That the conception of the surgeons is too inclusive is indicated by their disposition to describe the ligaments of the abdominal organs (for example, the broad ligaments of the uterus) as being formed of layers of peritoneum.

In the following discussion, therefore, the peritoneum will be regarded as being composed, first, of a cell layer; second, a basement membrane upon which these cells lie; and third, a connective tissue layer in which vessels and nerves lie.

It will be noted that in the general division above enumerated the peritoneum presents a series of layers analogous to those of the skin.

The Endothelial Cell.—The best summary of accepted knowledge regarding the structure of the endothelial cell is given by v. Ebner and may be freely translated as follows: "The free surface of the peritoneum is covered by a layer of polyangular, nucleated pavement epithelium, which average 22 microns in size, and whose surface presents a smooth, glistening surface."

The term "epithelial" as above applied to the covering cells of the peritoneum represents the common usage at the present time. When His first applied the term "endothelium" he had in mind a morphologic identity, which had its basis in his embryologic researches. The term itself was based on the belief that these cells lined all spaces which had been at no time connected with the external world. The cell surfaces included in this conception would be, besides those of the peritoneum, those lining the pleura, pericardium, the blood vascular system, joint cavities, and the meninges. Kölliker at first embraced this view of His, but after further embryologic studies had been made he rescinded his view.

Modern writers, as indicated in the definition of v. Ebner, above quoted, have reserved the term "endothelium" for those cells only which line the blood and lymph vessels and the anterior chambers of the eye, using the term "epithelium" for the cells covering the peritoneum and pleura. Waldeyer argues that there is a possibility that the peritoneal cells may be derived from the same cells as are the "external cavities," the gut tract, etc. The only argument for abandoning a term well established in usage, it will be noted, is that of a histogenetic possibility.

How uncertain is our knowledge relative to the origin of the germinal layer, from which the cells covering the peritoneum have been derived, can be appreciated by reviewing the literature. Tourneux et Herrmann, Neumann, Waldeyer, and Paladino were the first to depart from the nomenclature of His, all on embryologic grounds. Kolossow attempted to demonstrate morphologic grounds for his rejection of the nomenclature of His. He was consistent in that he proposed a like change in name for the cells lining the vessels.

Finally, the question of the existence of intercellular bridges, a problem still unsolved, has been emphasized as an argument in favor of employing the term "epithelium" for the cells of the peritoneum.

Numerous other arguments have been advanced for departing from the original conception of His. One is that in certain situations cilia have been noted (Dekhuyzen). These are said to occur chiefly in clefts and crevices. In answer to this it may be noted that, when a peritoneal surface has been irritated, an exudate ac-

cumulates on the surface. When treated with silver and sectioned this gives an irregularly jagged outline, which in a way resembles low cilia, but which may easily be demonstrated not to be such by means of Wiegert's fibrin stain. The deception is particularly liable to be imposing when two irritated surfaces lying in contact are pulled apart and then silvered (Fig. 1). It is interesting to note that those who found an important differentiating point in the fact that cilia had been noted on peritoneal cells (Dekhuyzen) overlooked the observation made by Van Beneden that in bats the uterine artery contains stratified cuboidal epithelium. I have reviewed the work of the last-named author, and the only tube I was

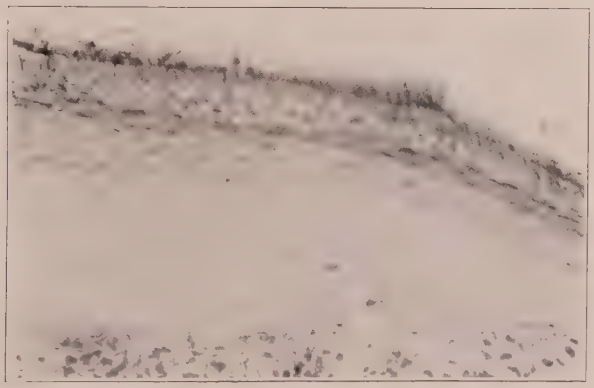

Fig. 1.—Cilia-like processes which were formed by forcibly separating two peritoneal surfaces after they had become agglutinated because of a slight chemical irritation.

able to find lined with stratified cuboidal epithelium in the pelvis of a bat, was in the ureter.

Others disregarded the alleged embryonal heterotopy, and counted the peritoneal cells with those of the blood vessels because of their close morphologic relationship. Prominent among these were Klein, Dekhuyzen, Ranvier and particularly Gegenbaur.

Marchand suggested the term "epithem" and proposed that the name of the region be added. Thus he would have "epithem" of the peritoneum, of the blood vessels, etc. While this author argues for the acceptance of Waldeyer's view, his suggestion of a new term would seem to be a confession of uncertainty in the embryologic classification.

If the old term "endothelium" is to be dropped, the suggestion of Minot to substitute the term "mesothelium" should be adopted. The development of the peritoneal structures from the mesoblast has been well established, and, besides, their activities in the stress of disease and regeneration are in line with other mesoblastic tissue.

Notwithstanding these arguments the term "endothelium" will be retained in its original meaning throughout this treatise; it has at least the justification of long usage, and is thoroughly established in the literature. What is still more important in their mode of reproduction after injury and their conduct in reactive processes these structures are not comparable to any other structures of epiblastic or hypoblastic origin. For these reasons they deserve a place in nomenclature all their own.

The Cell Outline.—In the early embryonal stage cells destined to become endothelium do not differ from those destined to become supporting connective tissue. They are at first cuboidal or globular, and gradually flatten out to the adult form. Toldt has well described these changes. My own observations in the human subject have shown me that at as early as six weeks the flattening out process is well advanced. Younger material was not available. At the eighth week the flat adult type is definitely reached though they are more regular in outline in the embryo than in the adult. The outline in all species of animals is very similar in these early stages. The peculiar variations characterizing the different species are not manifest until postnatal life.

Generally speaking, the endothelial cells are quadrilateral, presenting in a general way the oft-described mosaic. The outlines are irregular and undulating and the angles are often accentuated by being pushed between the diverging sides of adjoining cells, or obtunded by being impinged against a concave side of a neighboring cell (Fig. 2). The purpose of this variation in outline has never been determined. The irregularity is greater in the cells covering surfaces subject to successive contraction and expansion. For instance, they are most regular over the tendinous surface of the diaphragm and the inscriptiones tendinæ of the recti muscles, and are most irregular over the urinary bladder.

The cells, though varying somewhat in size, present a surprising uniformity when examined under like physical conditions. They

measure from thirty to fifty microns on an average, but may vary from twenty to ninety microns. Methods of preparation and fixation may cause an apparent variation in size. Cells prepared by being placed at once in strong alcohol, appear smaller than those in which the fresh specimen is stretched over a cork before fixation.

The endothelium of the peritoneum can be distinguished from that lining the blood and the lymph vessels by the form alone. The cells of the endothelium lining the blood vessels are eliptoid and arranged with their long axis parallel with the long axis of

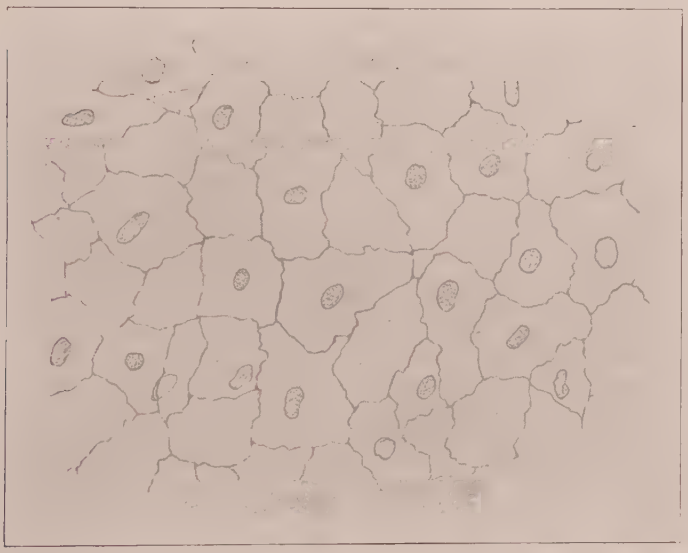

Fig. 2.—Normal endothelium of the peritoneum of a young rabbit.

the vessel, and are of quite uniform size. Those of the endothelium lining the lymph vessels are much more irregular in outline than the peritoneal endothelium and usually vary more in size.

The outline of the peritoneal cells also differs considerably in the various species of animals. The cells of reptilia show the most bizarre outlines, approaching in irregularity the endothelial cells of the lymphatics of mammalia. In this species there are to be found long projections of protoplasm, sometimes extending from one cell entirely across the diameter of two neighboring cells. These same processes have been described by Subbotin for the hu-

man subject. In the intensely silvered specimen such processes are closely simulated by portions of muscle cells lying below the endothelial cells, and I believe from the description of this author that he has been deceived by this appearance.

In the adult animals, along with the typical form of cell, there occur groups which in size and regularity of outline resemble the cells of the embryonic state. In some places these comprise groups of three or four, but sometimes larger groups extending over a considerable surface and comprising a hundred or more cells are encountered. These are noted particularly by Dybkowsky and Klein and Bizzozero. They were noted in the following situations: the abdominal surface on the diaphragm, the great omentum, the mediastinal pleura, and the tunica vaginalis, testis. These have been assumed to occur when a lymph channel lay especially near the surface. The generally accepted opinion has been that they represent young cells (Klein). The fact that they usually occur in some multiple of two constitutes almost the entire argument in favor of this view. Cells are sometimes found containing two nuclei, which in form and tinctorial reaction resemble those of the endothelial cells. (Compare Tourneux, Recherches sur l'epithélium des séreuses, J. de l'anat. et physiol., 1874, x, 66.) On cross section, too, they are seen to take the stain the same as the regular peritoneal cells. The most remarkable thing about these cell groups is their inconstancy. They are noted most frequently in batrachians. I do not remember ever having noted such a group of cells in an adult normal mammal.

More perplexing still are those areas similar in size to the above, in which the cells contain no nuclei. They react to stains just as the regular cells do, but no nucleus can be demonstrated. They are very irregular in their occurrence. Many preparations may be searched without finding any of these areas; and again, many may be found in a single specimen. It has been assumed that such areas are but instances where a part of the cell is exposed, while the remainder of the cell is covered by the surrounding cells. The difficulties in demonstrating this on cross section are manifestly so great that the fruitless results can hardly be wondered at. If they be accepted as cells in the process of taking their place in the regular layer, their origin has still to be ac-

counted for. Whether they are derived from the division of regular endothelial cells or from cells lying deeply, or whether they are artefacts, can not be stated. They are noted only occasionally in the lower orders, and apparently not at all in the human subject. It is my opinion that these areas represent cells whose nuclei, for some reason, failed to take the silver. I have never noticed such areas in specimens stained with a specific nuclear dye.

The disposition of endothelial cells to form a stellate group about a central opening is very remarkable, but its significance is not clear. They appear to inclose an opening at the center of the group. Klein regarded these openings as the true stomata. They are of rare occurrence, and are found chiefly in the frog. Their rare occurrence is sufficient to exclude the belief that they participate in any function necessary to the maintenance of life. The most remarkable thing noted by Klein is that the nuclei are located very close to the central opening. The theory was advanced that the nucleus is nearest that part of the cell where the greatest activity is going on. These cell groups were believed to warrant such a conclusion. This argument is negated by the fact that when such specimens are subjected to nuclear dyes, nuclei are demonstrated in their usual position and usual form.

The special types of cells above mentioned probably have no significance. Their rare occurrence has been noted. As our technic improves they become progressively less. I have observed none for many years, despite a diligent search in order to subject them to the scrutiny of more mature experience.

Under certain conditions the cell outline may undergo a change. It is particularly to be noted that the cells covering the pregnant uterus are elongated, and in no wise resemble the cells of the non-pregnant uterus. They resemble more those lining the blood vessels except that they are still more spindle form. Sometimes cells covering pathologically enlarged organs may be elongated in the same manner though less markedly. A gradually expanding organ in a state of hypernutrition seems to present the conditions upon which these cell changes depend.

The Nucleus.—The nucleus of the cell is ovoid (Fig. 2), usually measuring from eight to twelve microns in width, and from twelve to sixteen microns in length. It is usually situated near the cen-

ter of the cell, but is sometimes somewhat eccentrically located. Its long axis always coincides with the long axis of the cell. Rarely two nuclei may be observed in the same cell. A nucleolus usually occupies one of the foci of the ellipse; more rarely two exist. The nucleoli stain an intense uniform hue with nuclear stains, while the nucleus stains with a granular appearance, usually fine in character, though sometimes lumps of considerable size are observed.

A more complex structure of the nucleus has been recorded. These observations were made with the cell *in situ*, usually in specimens prepared with silver. Structures beneath the cells can not be distinguished from intracellular structures. Channels have been described as extending from the nucleus to the protoplasm. Elastic fibrils sometimes remain unstained, and may appear as channels within the cell protoplasm. Such confusing pictures are not observed when the cell is isolated and then stained.

The Cell Protoplasm.—The extranuclear cell protoplasm examined in the fresh state is transparent. Stained with protoplasmic dyes it presents a uniform substance. Treated with silver nitrate it becomes finely granular, but the granules are less abundant than in the nucleus, due no doubt, to the lesser chloride content of this portion of the cell.

The channels mentioned in the preceding paragraph have been depicted extending into the protoplasm, forming a more or less complex network (Fig. 3).

The Cell on Cross Section.—The definition of Klein gives a good presentation of the cell as seen on cross section. It may be freely translated as follows: "The cells are quite flat, and contain a roundish or oblong nucleus, which exceeds the cell in thickness, so that the protoplasm lying about it is bulged outward." This emphasizes an interesting and characteristic feature that has been entirely overlooked by some of the later investigators, namely, the manner in which the cell is made to take a spindle shape on cross section.

The cell varies from four to eight microns in thickness in the extranuclear portion. The body of the cell in a given preparation is of strikingly uniform thickness in all parts. In the region of the nucleus where the cell becomes thicker to accommodate the relatively thick nucleus, the relative proportion of protoplasm lessens.

The nucleus is from three to six microns thick, being less in this diameter than in the diameter parallel with the surface of the cell. In young endothelium, and in that covering the viscera where the cell body is thicker, the bulging of the nucleus above the remainder of the cell is not so apparent.

The thickness of the cell varies greatly in abnormal conditions. It is very sensitive to irritants, responding to them by an increase of the volume of the extranuclear protoplasm.

The cross section of the protoplasm of the unstained cell shows the same glistening, homogeneous appearance as seen on the flat surface. The cell protoplasm takes the acid dyes very intensely, resembling in this respect the superficial cells of the epidermis. The free surface takes these stains more deeply than that portion

Fig. 3.—Klein's idea of the canalization of the cell protoplasm. (Redrawn from Klein.)

of the cell nearer the basement layer. When the cell is irritated these acidophilic properties are lost in a large measure.

A number of observers have recorded what they believe to be peculiarities in cell structure. Subbotin studying blood vessels concluded that the cells overlap like shingles on a roof. These observations have never been confirmed. Bizzozero and Salvioli noted processes extending some distance beneath the neighboring cells, sometimes exceeding in length the diameter of such cells. That this observation is an error can be easily observed in deeply stained specimens. The processes they noted were very obviously heterogeneous cells, mostly nonstriated muscle cells lying below the peritoneum.

Cilia.—From time to time the presence of cilia on the peritoneum endothelium has been described. Paladino first described

them. Kolossow described them at length. Muscatello confirmed this opinion. V. Brunn in ignorance of previous work, recorded them as a new discovery; and in a later paper he reaffirmed this opinion. These so-called cilia are described by Kolossow as very delicate, short processes having a length of two microns. He found them everywhere in a large variety of animals on the pleuroperitoneal surface, except on the great omentum. In lower animals, fishes, reptile and amphibians, he found no trace of them. He was able to demonstrate them only on section, failing to do so by physiologic tests. V. Brunn found these structures with great regularity, but not invariably. He ascribes failure to find them in all cases to their great delicacy, believing them to have been lost during the process of preparation. He found them on inflamed surfaces, as well. He does not believe that they possess the usual functions of cilia, but ascribes to them the function of increasing the rate of absorption. He noted that they increase in length by irritation, and he concluded that they have retractile capabilities.

In my studies by means of fibrin stains it was possible to demonstrate that these so-called cilia were nothing more than fibrin in deposits due to coagulation of fluid normally covering the surface of the cells. They may be demonstrated best by staining slightly inflamed membrane with silver. It then appears as if a fibrinoid exudate as it exuded from the cells and intercellular clefts became stained by the silver. By careful study with the high-power instrument these cilia are found extending from the intercellular lines, as well as from the surface of the cell. In fact, sometimes the "cilia" are seen to be longest over the intercellular lines. An unusually well developed example is presented in Fig. 1, produced by gently irritating the peritoneum of a mouse and then staining lightly with silver.

V. Brunn partly confirmed this idea when he noted that they are increased in length by irritation of the surfaces. By gently rinsing the surfaces to be studied with some indifferent fluid before fixation, these objects are not found. In substantiation of his belief, v. Brunn quotes a number of writers who found cysts lined with ciliated epithelium in the liver, and Pick, who reported an adenocystoma of the pelvic peritoneum, which was lined by ciliated epithelium, which he regards as having been derived from

the peritoneum. In regard to the latter, proof is not altogether satisfactory, it being impossible to exclude origin from some of the fetal structures in this region.

The Intercellular Cement Substance.—A special cement substance between cells was first described for the nervous system by Koch and Schiefferdecker, who regarded it as a mass which filled out the spaces between the Lantermann's processes. Johansson attempted to demonstrate this substance by a special process. Rabl-Rueckhard and Schweigger-Seidel and Dogiel regard the fluid between the cells as lymph. Flemming and Merk, on the contrary, noted that the intercellular substance differed from the fluid in the lymph vessels.

My own researches on this problem lead me to the same conclusion as that of the last-named authors. The substance between the abutting cells differs markedly from that below the cells in its capacity to reduce silver.

The difference is sufficiently marked to warrant the term "cement substance" for the substance between the cells, in contradistinction to the "ground substance" for the fluid lying between the fibrils, as was suggested by Waldeyer. That the former is acidophilic and the latter basophilic I have not been able to determine. The cement substance when precipitated with silver produces a much darker deposit, due to the larger size of the granules. This probably is due to the greater chloride content of the former substance. At any rate the appearance of each may be imitated by employing solutions of chlorides in albumin of varying concentration.

It is of interest to speculate on the physiologic differences of these substances as observed in the endothelium of blood vessels. The substance between the cells is, of course, exposed to the blood stream, but does not produce coagulation. If, for any reason, the intercellular connection is broken the ground substance from beneath the cells comes in contact with the blood, coagulation takes place at once. The same is true of peritoneal endothelium. The intercellular substance does not invite adhesions, but, when this is removed and the ground substance gains the surface, adhesions take place. Every attempt on my part to arrive at a more exact conclusion has failed.

An analogy has been drawn between the cement substance beneath the endothelial cells and the ground substance between the muscle cells. The substance between the muscle cells reacts to silver and to blood coagulation, somewhat as does the ground substance below the endothelial cells, but the action is much less intense. There may be an analogy, but not an identity.

Relative to the chemical nature of the cement substance, but little can be said. Rollett compared it to a colloid. Eichwald compared it to the "musin" in ovarian cysts and goiters. Schaffer regards the substance as a mucin. Unfortunately, no studies of this substance have been made since the development of modern colloidal chemistry.

Intercellular Processes.—The hypothecated existence of intercellular processes has but recently come to play a part in the discussion of the histology of the endothelial cell. Several unimportant references to the possibility of the existence of such processes are found in the older literature. Hammar described protoplasmic bridges in the blastomere. Their ultimate fate was not followed. It remained for Kolossow to renew the discussion. No paper since has contributed anything to the subject, nor has confirmation of his researches been forthcoming. Nevertheless, recent authors have accepted his conclusions without question.

The study of the problem of the presence of intercellular processes may be approached in several ways. Theoretically, the hope existed that, if present, they might be demonstrated by staining the cells with a specific protoplasmic dye, as is done in epidermal cells; but this hope has not been realized. All such studies have been negative. It is only by the use of a chemical that forms a precipitate with the cement substance between the cells that an interrupted line can be produced under certain conditions. Kolossow assumed that the interruption was due to the presence of intercellular processes (Fig. 4). In the absence of cells the cement substance, when treated with silver nitrate or osmic acid, forms globular masses, just as it does when the cells are present. The spaces between these globular masses resemble the bridges shown by Kolossow,—bridges without cells, as it were. This occurs only when osmic acid is used. When the more concentrated solutions of silver are employed an interrupted line is produced, but this

can be penciled out, and the edge of the cell is seen to present a smooth outline. When some of the newer organic preparations of silver, notably the tannate, are used, such an interrupted line does not occur. The same is true when weak solutions of the nitrate are used. Cells mechanically torn from their bed do not show a dentated outline.

The attempt to compare the intercellular bridges of the endothelium with those between the muscle cells is not apropos. Moreover, more recent studies (Schaffer; v. Lenhossék) have shown that

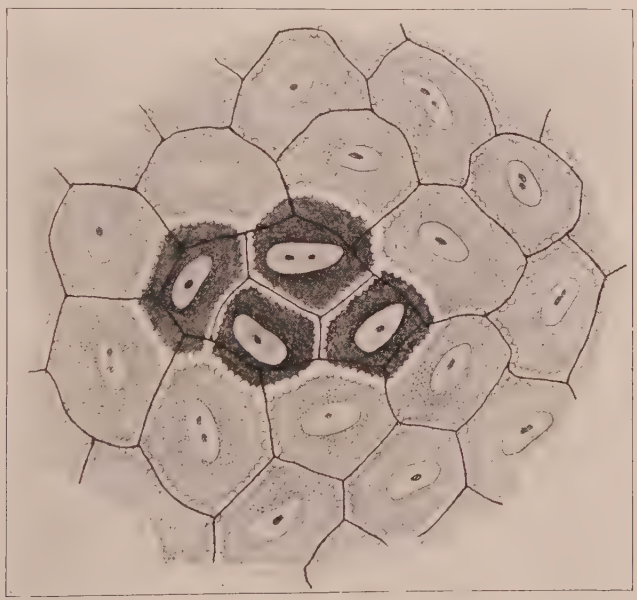

Fig. 4.—The dentated borders of the cells represent Kolossow's idea of intercellular bridges.

what appeared to be bridges between the muscle cells was merely cross sections of fine connective-tissue bundles surrounding the muscle fibers. Hensen also calls the fluid surrounding these small fibers cement substance (Kittsubstanz).

The connecting fibrils described by Nicolas as extending from the endothelial cells to the underlying connective tissue are obviously artefacts due to the use of silver nitrate. The substance in which these artefacts are formed is colloidal, and may be given the term suggested by Waldeyer, namely, cement substance. This

substance is relatively more abundant in the batrachians, upon which Nicolas made his observations.

A more complicated cell structure has been described by a number of authors. Tourneux noted two strata in the peritoneum of batrachians. The upper layer was homogeneous, while the lower layer was formed of granular protoplasm, according to him. This error is easy to make. When a mesentery is viewed both layers may fall into the field of the microscope at once. Or one endothelial surface and one wall of a wide lymph channel may fall into view. Special stains of the connective tissue may be required to prove that the two endothelial layers are not actually superimposed.

Kolossow made an exhaustive study of this subject, and concluded that the endothelial cells are composed of two layers, the cell proper and the cover-plate (Deck-platte). He thinks the stomata occur between the cover-plates and that intercellular processes are to be found between the cells proper.

My researches with cross sections of tissue treated with osmic acid according to Kolossow's technic have shown that the precipitate extends through the whole thickness of the cell. Any channel that may exist must be below the cell.

The importance that Kolossow places on the ability to determine the position of an object by the focusing of the microscope is hard to understand. Focusing on these lines, even with the most perfect adjustment, causes a kaleidoscopic change in their appearance when viewed with the high power. It is only by making cross sections that one can gain any accurate knowledge of the relation of the various structures.

The studies that Kolossow made of cross sections of the tissues are not convincing. The nuclei, which he regards as those of leucocytes, are much like those of endothelial cells. The resemblance is too close to permit of a differentiation. Of course when tissues are impregnated with osmic acid the finer cell structures are much less distinct than when stained with other methods.

This method confuses the cell with the subendothelial connective tissue and fluid surrounding it. The results obtained by this author are not obtained when the isolated layer is studied, nor can such a cell composition be demonstrated by any other technic.

I am forced to conclude that the appearance of the cell is due to the technic.

Kolossow assumes that silver does not produce these broad lines because it does not penetrate deeply enough. When the peritoneum is irritated before applying the silver, or when a hot solution of silver is used, lines as broad as those produced by the osmic acid appear. The reason for the appearance of broader lines when osmic acid is used would seem to be due to its more intense power of reduction when in contact with the cement substance, just as silver nitrate produces a broader line than silver tannate.

The Stigmata and Stomata.*—The stigmata and the stomata have played such an important part in the physiology of the peritoneum, as well as in the physiology of absorption and in the process of inflammation, that an extended inquiry into their structure and significance is imperative. That they still dwell in the minds of investigators is demonstrated by the papers of Buxton and Torrey. To the use of silver nitrate as an agent in microscopic technic is due the whole controversy about the stomata. It was Coccius and Flinzer who first used the nitrate of silver to demonstrate the cell outline of Descemet's membrane. This method was perfected by His, who first noticed the occurrence of small dots and rings in the intercellular lines. The discovery was accidental, for, as the title of his paper indicates, he was concerned only with a review of v. Recklinghausen's work. Oedmansson himself hardly regarded the discovery seriously, but was rather inclined to believe that the rings were real openings because of their regularity. He regarded the stigmata as deposits of silver albuminate because of their irregular size and form. Not until after the same structures were observed by His in the lymph vessels was Oedmansson's discovery regarded as of importance. From that time on the belief in their identity gradually became general. A number of other papers were written about this time, the most important of which were those of Ludwig and Schweigger-Seidel, Dybkowsky and Schweigger-Seidel and Dogiel, none of which added greatly to our knowledge of the subject in advance of that presented in previous publications.

*The following account is taken almost verbatim from my paper, "The Morphogenesis of the Stigmata and Stomata Occurring in Peritoneal and Vascular Endothelium," Tr. Am. Micr. Soc., 1901, xxii, 63.

The highest interest in the stigmata and the stomata was not reached until Cohnheim described them in the blood vessels, and used them as important elements in his theory of inflammation (v. Recklinghausen). It is to this fact no doubt, that these structures owe much of their prominence, for, after their acceptancy by Cohnheim, their existence was almost universally regarded as proved, though no additional evidence had been adduced to strengthen the theory.

That the existence of these structures as morphologic elements was never established upon a sound histologic basis may be gathered from the fact that very soon after they were first described there were not wanting investigators who regarded them as artificial products, due to the reagent used. Among the earlier writers to express this opinion may be mentioned Auerbach, Afonassiew, Foà, Klein, and Schweigger-Seidel. The last two authors regarded them as real openings, but declared against the function ascribed to them on the ground that no opening could be demonstrated in the basement membrane (membrana limitans), without which the stomata would necessarily be functionally useless. Later Klein seems to have changed his view somewhat. Klein and Smith ascribed more importance to the stomata even going so far as to classify them into two groups,—stomata vera and pseudostomata. In the former group they reckon only those occurring in the center of groups of radiating cells (Fig. 5). To the list of opponents must be added Tourneux. He is very emphatic in his opposition, declaring without qualification that they are artefacts. He based his argument on the fact that starch and carmine granules are not absorbed when an emulsion of them is introduced into the free peritoneal cavity.

These papers were written about the time Cohnheim announced his theory of inflammation. So brilliant were his discoveries and so striking his explanation of the phenomena he observed, that the arguments of the opponents of the theory of the existence of special openings between the cells were futile. It seemed very clear that the leucocytes, and, above all, the red corpuscles, in order to escape from the blood vessels, required the existence of openings that would allow of their escape. The discovery of Oedmansson was early seized upon as offering the needed explanation.

Arnold, by his careful researches, aided greatly in strengthening the position of Cohnheim's theory, and more particularly the idea of the passage of the blood cells through preformed openings. It was he who first observed the blood cells actually to pass through the preformed openings, thus apparently adding demonstration to the theory. Arnold presents a cut showing a leucocyte in the act of passing through one of these openings. Lavdowsky likewise described the escape of leucocytes and illustrated their method of escape.

Notwithstanding the reputation of these authors, doubts began to arise as to the correctness of the observations, mostly perhaps on account of the extreme difficulty of excluding error in deter-

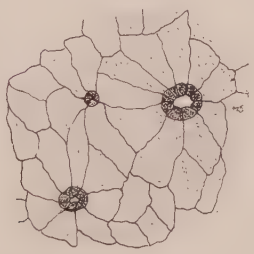

Fig. 5.—A stoma formed at the point of junction of many cells. (Redrawn from Schweiger-Seidel and Dogiel.)

mining the exact point in the vessel wall at which the blood cell made its escape.

Another paper that has much weight in influencing opinion is that of Muscatello, who approached the subject largely from the physiologic side. He ascribes to the stomata the office of absorption, but declares that they exist on the centrum tendineum of the diaphragm only. Ranvier advanced a very convincing argument against the stomata when he showed that their occurrence could be much reduced in number by first rinsing with distilled water the surface to be treated with silver solution. He also added the additional important argument that their irregular distribution counted much against their functional importance.

Whatever opinion one may hold as to the cause of it, the fact remains that the stomata no longer play the part they formerly did in the explanation of physiologic and pathologic processes.

Adami regards them with skepticism. This fact must be interpreted to mean that the structures have been abandoned, and the subject no longer offers a fruitful field for investigators. The decadence of the stigmata and stomata has been due to the fact that, with the shifting of the theories of inflammation, they have become less necessary. The lessening of their importance is not due to any new histologic studies into their nature.

That this neglect has not been altogether universal is shown by several recent papers dealing with the histology of the peritoneal endothelium. The more elaborate and complete of these is by Kolossow, in which he restores the stomata to their former position.

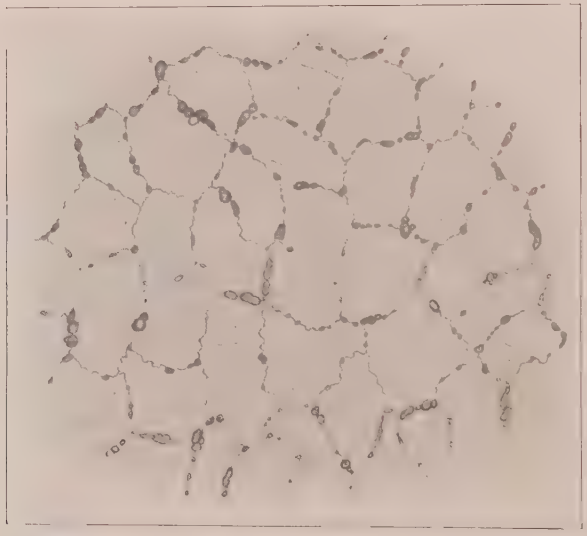

Fig. 6.—Classical stigmata and stomata produced by treating the peritoneum of a rabbit with a 1 per cent solution of the nitrate of silver.

Kolossow makes a modification, in that he believes the stomata occur between the cover-plate (Deck-platte).

In a more recent paper Ussow takes a similar position. He believes that, ordinarily they do not exist, but are brought about by the contraction of the cell protoplasm. In the short abstract of his paper which I have seen, no reasons are given for this conclusion.

The other side of the question has recently been strengthened by a very clear paper by Meyer. He concludes that the stomata

are not real openings, and he arrives at this conclusion because of their irregular distribution and because of their irregular size and shape. He believes, however, that they may be artificially produced by mechanical means, though he ascribes no specific importance to this fact.

Rawitz takes a delightful middle ground by saying that, if stomata do not exist, there are at least "soft places."

Nearly all investigators have used the same method, which consists, briefly, in treating bits of excised tissue with a solution of silver nitrate and then exposing them to the sun until they become brown. Usually a one per cent solution has been used, as first advised by His. Ranvier alone used a weaker solution (1:300 to 1:500) and for injection as low as 1:800. To this modification in technic may well be ascribed the superiority of the results obtained by him. Alferow used instead of nitrate of silver, salts of the organic acids, silver picrate and lactate. By this technic he obtained large areas without stigmata and stomata. The superiority of this technic, unfortunately, was not recognized.

The stigmata are small brownish-black points, varying in size from a fraction of a micron to ten microns. They are usually irregular spheroids, but often angular and are occasionally elongated ovoids, occurring at irregular intervals in the course of the intercellular lines between the endothelial cells, when a serous membrane is treated with silver nitrate and exposed to the sun (Fig. 6). If the peritoneum is irritated before applying the silver solution the stomata are larger (Fig. 7). The stigmata have received little attention from writers, and they need not be further considered. It may be added that some writers regarded them as incipient stomata. By stomata are meant the brownish rings, ovoids, or ellipses, occurring under similar conditions as mentioned above, situated anywhere in the course of the intercellular line, but found most typically at the point of junction of three or more cells.

Those who accepted the stomata theory engaged in a controversy as to their genesis. Some regard them as preformed, others as due to the passage of leucocytes, but the majority regard them as due to the stretching of the membrane. The cells were believed not to be quite big enough to cover the entire surface when the basement membrane is put on the stretch. Kolossow worked out this point

and attempted a direct demonstration of the theory. He did this by stretching a serous membrane across the end of a glass tube, and then bringing about various degrees of distention. He found that the stigmata and stomata increased in number as the distention was increased, and in direct proportion. I have made a long series of experiments to determine this point. The result was invariably that, even when the membrane was sufficiently stretched to produce a solution of continuity of the cells, they did not separate in the intercellular lines, but tore across the cell in the great majority of cases, the direction of the separation depending on the line of rupture of the basement membrane. The cells do not

Fig. 7.—Heavy precipitates in the intercellular lines. (Redrawn from Oedmansson.)

separate as long as this membrane is intact. Even if by chance the cells do separate in the intercellular line the separation does not occur at certain points, but occurs in a straight line.

The theory of their formation by the leucocytes was abandoned on the ground that, when a vein is tied in which diapedesis has begun, it ceases. If due to the activity of the leucocytes the process (of stomata-formation) would go on. Muscatello, however, still adheres to this view.

If, in the study of the genesis of the stigmata and stomata, the classical method of treatment, with nitrate of silver and exposed to light, be followed, the basis of the whole controversy can be

readily observed, for all the different pictures described by the various investigators may be made out. In reviewing the results the most striking observation is that of Ranvier, already referred to, namely, that the number of stomata may be lessened and for large areas prevented by the preliminary rinsing with distilled water. So striking is this fact that the probability at once suggests itself that these structures are due to some factor that can be removed with distilled water. That actual openings could be thus removed, of course, no one would for a moment argue. That large areas may thus be obtained free from the stomata is a fact vouched for by various observers. Why the cells in so large areas should occur without the ability to perform the same function as identical cells in other localities perform, is a question that Ranvier asked years ago, and that still remains unanswered. Observers are likewise agreed that exposure to the sun increases the number of both stomata and stigmata. The same is true if the membrane is covered with an excess of lymph. Oedmansson correctly states that the lines are broader if the strength of the silver solution is stronger, or is allowed to act for a greater length of time. He also correctly observes that it is due to the action of the silver on the cement substance, and not due to its action on the edge of the cell, for the lines may be penciled out. He might have added that this in no wise influences the distance between the edges of the cells, for the increase in width of the line is due to deposits on top of the cells.

The effect of light is a factor that has been systematically studied. Handling the tissues in an entirely dark room is difficult. An attempt to solve this problem led me to develop a new mode of using silver. Knowing that Ranvier had injected silver solution into the adipose tissue in recently killed dogs, in order to outline the fat cells, I injected silver solution into the peritoneal cavity, in order to secure the reaction on the peritoneum *in vivo*. The effect of light may thus be determined with a certainty. The intercellular lines in preparations so made are seen to be present, and thinner and more regular than after the usual method, and stigmata and stomata are not to be found.

The injection enabled me to test the distention theory, already mentioned, as being held by a goodly number of observers. By

injecting a large amount of dilute solution into the free peritoneal cavity any degree of distention can be produced. This would serve only, of course, if the endothelium of the abdominal wall were the object of study. If it is desired to study the endothelium covering the intestinal canal, a small amount of the solution may be injected into the abdominal cavity, and the intestine then distended with air. After many trials under the most varied conditions, it can be stated that stomata do not occur under these conditions. This method of testing the distention theory would seem an ideal one, for the tissues are uninjured in any way and there is no escape of blood or lymph to obscure the results.

On account of the influence the form of the cell was believed to have on the production of stomata, it will be proper to consider the effect of a simple distention on the cell outline. The distention experiments before mentioned show that the cells are large enough to cover the basement membrane without changing their form to any appreciable extent. When a serous membrane has less surface to cover, it forms folds and the individual cells do not contract.

Another method of determining the effect of distention may be briefly mentioned. An abdomen may be distended with air, and the intercellular spaces made apparent by painting the surface of the abdomen with solid sticks of silver nitrate. The cell outlines are brought into view. No influence on the production of stomata can be observed.

Still another way of demonstrating the absence of openings between the cells is as follows: a bit of the abdominal wall is stuck to a slide, the endothelial surface down. Afonassiew's method was used. After firm adhesion has taken place between the tissue and the glass slide, the membrana limitans, with all the other tissues, is removed, leaving the endothelium alone adherent to the slide. It may now be treated with any desired stain, preferably fuchsin. The edges of the cells will be found to lie closely together without the existence of openings. The last method gives some very satisfactory results, though the method is somewhat difficult to employ. For some time I have been experimenting with iron and tannin for bringing out the cell outlines. By this

method, likewise, no stomata appear, nor are the cell outlines affected by stretching.

Besides the stomata above described similar objects situated elsewhere than in the intercellular line have been described and illustrated. These have been described in every conceivable place that silver can penetrate. In many illustrations they are depicted scattered promiscuously over the cells (Fig. 8). Oedmansson's original publication was the first to present them, but they may be seen in many productions since that time. It remained for Meyer to recognize the true conditions. Fig. 10 in his paper presents the appearance very well. These same structures may be seen lying upon the endothelium of the lymph vessels. They may often be seen in the body of the cell, frequently forming gyrated figures.

Fig. 8.—The stomata appear over the body of the cell away from the intercellular line.

Again the circles may occur in the intercellular line, but through which the line continues uninterrupted. This must be distinguished from objects occurring below the endothelium. These stomata may be seen below the endothelium between the muscle fibers. This may, instead of forming rings, form solid patches, thus resembling a cell. Meyer very properly asked "If they are to be called stigmata in one situation why not in the other?"

In rare intervals cells radiating in a peculiar manner from a central point are observed (Fig. 5). A few writers, notably Klein, regard these as the only true stomata. Their significance is unknown. Because of their rarity they can have no important function. I have never observed them except in batrachians, and in these animals only at rare intervals.

Sometimes small cells are observed between the larger ones.

Ranvier regarded these cells as guards for the stomata, filling them up valve-like. When impregnated with silver they have been described as stomata.

Cells above the endothelial cells are but rarely found. For a long time, I observed cells in this situation, believing them to be endothelial cells which were contracted from irritation, as described by Ranvier. On cross-sectioning the tissue the endothelial layer was found to be intact. They are round cells, notably present in the peritoneal fluid, which have become attached to the endothelial cell in response to some irritation.

The first observer to note the occurrence of small cells between the larger ones was Dybkowsky. Klein advocates the view that they were cells in the course of development. Ranvier regarded them as leucocytes filling up the stomata. Virchow has been incorrectly quoted as advocating this view. As a matter of fact, the statement was made that "multinuclear cells occur below the epithelial cells of the peritoneum." These words were written before the controversy began and before it was known that leucocytes occurred outside the blood vessels; indeed, it was in support of the theory that they do so occur that the statement was made. The leucocytes are really the only ones that ought ever to have been mistaken for stigmata or stomata, for, as Tourneux and Herrmann pointed out, the oblong nuclei and the bright nucleoli are always sufficient to distinguish the other cells occurring in this locality from them. This discussion could have arisen only from a failure to study them in cross section.

The exact significance of these cells is not pertinent to the subject; it is sufficient for the purpose to show that they form a part of the regular cell layer.

It is the cells located below the endothelial cells that have been mistaken most frequently for stomata; therefore they are of greatest interest in this connection. Many examples of this confusion may be seen in the literature, notably in the publication of Oedmansson. Kolossow classes them all as leucocytes. As already mentioned, Stöhr pictures several as connective tissue cells. Both kinds do no doubt occur in this place. There are still others occurring here, probably belonging to the clasmatocytes of Ranvier or to those large cells which are now regarded as taking some part

in the process of inflammation. It is sufficient to say that they are all located below the endothelial layer.

By the use of a 1:1000 to 1:500 solution of nitrate of silver injected into the peritoneal cavity of animals *in vivo* one secures preparations in which the intercellular lines are perfectly formed without the appearance of a single stomata. I have repeated this many times on various species of animals.

By careful technic, as above mentioned, it is easily demonstrated that the stomata are spurious products formed by a precipitate of silver. A determination, if possible, of their exact chemical composition seems very desirable. It has been universally accepted that the intercellular lines are formed by the action of the silver nitrate upon the albuminous cement substance, forming an albuminate of silver. This assumption, advanced by His has been accepted by all writers. In approaching this subject I thought to prepare myself for the investigation by studying the chemical nature of silver albuminate. I was surprised, after spending some time in a literary search, to find no mention of silver albuminate in the most extensive works on chemistry. Surprise rose to amazement when one of the ablest chemists was appealed to for aid, and his reply was that he knew of no such substance; in fact, he stated that such a substance was a chemical impossibility.

Returning to the observation of Ranvier,—that the occurrence of stomata could be prevented by rinsing the membrane before applying the silver,—it was quite natural to remove some of this fluid for examination. Some of the fluid normally covering the free surface of the peritoneum was carefully transferred to a slide, and exposed to the action of a very dilute solution of silver nitrate. Minute droplets of this mixture were then transferred to a clean slide, and placed under the microscope. What were clear droplets became, on exposure to the sun, dark brown rings. The rings produced by evaporating small drops of silver and lymph, or whatever the composition of the substance removed may be,—bear a striking resemblance to the stomata produced in the regular manner. The pictures so produced are so striking that the statement seems warranted that stigmata and stomata may be produced independent of a serous membrane. This statement is equivalent to

saying that the stigmata and stomata are the product of the action of a solution of silver nitrate upon peritoneal fluid.

In order to test the silver chloride theory of formation of stigmata and stomata, the following experiment was performed: 5 c.c. of a sodium-chloride solution was injected into the free peritoneal cavity of a mouse. After a few minutes a dilute solution of silver nitrate was injected through the same needle. After the silver solution had had time to mix thoroughly with the sodium solution, the needle was withdrawn, the opening made by the needle ligated, and the animal given the liberty of the cage. Presumably, the silver was precipitated as soon as it passed the point of the needle, although absolute proof of this is difficult to produce. The remarkable fact was noted that when the silver solution was preceded by a sodium-chloride solution the lines did not appear until from thirty minutes to three hours afterward. In either case the appearance of the lines is the same. The difference was noted that, when the silver solution was preceded by a sodium-chloride solution, these always appeared precipitated over the body of the cell, which is not the case when the silver is used alone. An attempt was made to change the reaction of the albuminous substance covering the peritoneum before injecting the silver solution. The means employed to accomplish this were to inject a solution of equal parts of 0.1 per cent of hydrochloric acid (c.p.) and distilled water, followed after a few minutes by the silver solution. The lines appeared. It was interesting to note that the endothelial cells stood this severe treatment with little injury; also that, when so treated, the intercellular lines were very fine, with no broadening whatever.

Stigmata and stomata were produced by mixing egg albumin with silver-nitrate solution upon a cover-glass. As an example of the striking figures produced by the action of the silver nitrate solution on an albumin solution Figs. 9 and 10 may be referred to. The specimens from which these drawings were taken were made by mixing a drop of 0.5 per cent of silver nitrate with a dilute albumin solution, and gently warming the solution and allowing it to evaporate with the result noted in the figure.

The exact composition of the product of the silver and albumin can not be stated. The fact that the fluids of the body contain

0.65 per cent of sodium chloride together with the well-known fact that the nitrate of silver in solution is more sensitive to chlorides in loose combination than to any other substance, has caused me to accept as a working hypothesis the assumption that the

Fig. 9.—Figures resembling stomata produced by nitrate of silver and a dilute albumin solution.

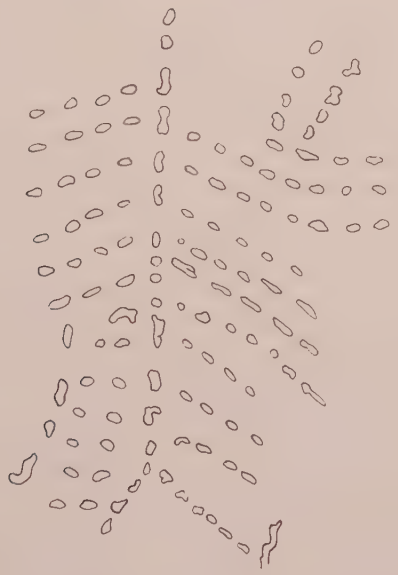

Fig. 10.—Figures produced by the same technic as the preceding figure.

substance is a chloride of silver, perhaps holding an albuminous substance as an admixture.

Later researches indicate that it is a calcium chloride, instead of, or in conjunction with, sodium chloride, that acts in reducing

the silver. Any substance that lessens or destroys the calcium in the fluids prevents the formation of the rings. However this may be, the validity of the silver-chloride hypothesis is not lessened. This assumption has to support it the following facts: (1) no such substance as silver albuminate is known; (2) only when a chloride-containing substance is present are the rings formed; (3) the well-known sensitiveness of the silver nitrate in solution to chlorides in loose combination; (4) combinations of silver with nuclein are known, but nucleins are not contained in the substance in question.

Special Openings in the Diaphragm.—Special openings in the diaphragm have received a good deal of consideration in the past. It would hardly be worth while to give it serious consideration, had not the alleged presence of such openings given rise in recent years to theories regarding the proper treatment of peritonitis. The presence of those openings was suggested by von Recklinghausen's experiments whereby it was proved that milk is absorbed by the diaphragm, and by Ludwig and Schweigger-Seidel who proved that after evisceration Prussian blue may be absorbed by this route. Because of this fact special openings seemed necessary to admit of their passage. Special openings are accepted and figured (copied from Ranvier) by Branca. These openings are declared to be pits in the peritoneum which communicate with the interstitial spaces in the diaphragm. Wandering cells are figured as arranged about these orifices, which are believed to be direct openings between the peritoneal cavity and the connective-tissue spaces. The connective-tissue spaces at that time were supposed to be a part of the lymphatic system. The approximate number of these openings was never estimated. I have never seen such an opening in a specimen stained *in vivo*. Such openings are never discovered by this method; nor are they seen when the diaphragm is stained on the stretch. They are found only when the diaphragm is contracted, thereby causing a folding of the peritoneum. These pits may be made to disappear by a cautious stretching of the membrane. The endothelium covering the diaphragm is the same as that covering the parietal wall elsewhere; and difference in the size of the cells is due to the contractility of the organ, the cells being smaller where the organ contracts. This is less likely to occur over the centrum tendineum than over the muscular part, provided fresh

centrum is examined. If the centrum is hardened in fluid, causing marked contraction of the fibrous tissue, the reverse may be the case.

Not only is the endothelium covering the diaphragm identical with that in other parts of the peritoneal cavity, but the underlying lymphatic vessels are also identical. They are not different in structure, nor are they more abundant. They are never connected with the peritoneal surface by special openings or otherwise. Even in instances in which solid bodies gain the lymph stream, there is no evidence that they were absorbed through the surface of the diaphragm. That the diaphragm offers resistance to the absorption of solid particles is made manifest by the rarity of the transmission of infection from one surface to the other.

The Basement Membrane.—Immediately below the endothelium is a complete layer of connective-tissue fibrils. This membrane was first described as the membrana limitans by Bizzozero. When specimens of peritoneum, endothelial surface down, are dried upon a slide, and the overlying tissues are stripped off, the basement membrane may be obtained as a complete layer. Specimens so prepared are composed of parallel bundles of fibers, together with the cement substance in which they are imbedded. This cement substance, as well as the fibers themselves, takes the connective-tissue dye. The acidophilic properties are exhibited only in direct specimens. When so prepared there results a homogeneous sheet of intensely stained tissue. When carefully prepared, there are no openings. When the tissues are dried or hardened in alcohol before staining, openings may appear here and there in the preparations. Some of the older writers held that it was by virtue of these openings that absorption was possible, and that regions in which no openings were found were not capable of permitting the passage of fluid from the peritoneal cavity. Their occurrence is too rare to warrant us in ascribing to them any functional importance, and it is my opinion that they are artefacts.

When tissue is fixed in alcohol and mounted in the regular way, the cement substance in which the fibrils are imbedded is dissolved out and the membrane is seen to be composed of a network of fibrils, the strands of which are arranged parallel to each other. This indicates that, in the unfixed specimens, it is the semifluid

interfibrillar tissue which, taking the stain as intensely as the fibrils, gives the layer the appearance of a solid sheet of tissue. This layer bears, it seems, a close resemblance to the similar layer in the kidney as described by Mall. The substance lying between the fiber bundles has the properties of a cement rather than those of a membrane.

When a bit of peritoneum is slightly macerated (HCl, 0.1 twenty minutes) and then silvered, the endothelial cells are loosened and are readily penciled out. The cement between the cells and the basement membrane remains, and is made apparent by the silver. Membranes that have been silvered in the early stages of inflammation, give the same picture. When viewed on the flat surface a membrane so prepared gives the appearance of a tiled floor from which the tiles have been removed, leaving the cement behind still impressed with the depressions recently occupied by the tiles. Basement cement substance would be a more accurate designation, and would serve to separate it in conception from the connective-tissue layer, which is properly called a membrane. The whole reminds one of a tile floor laid on a wooden floor. The connective-tissue fibrils are represented by the boards in the floor. The cement substance lying between and upon the connective-tissue fibrils is represented by the boards in the floor. The cement substance lying between and upon the connective-tissue layer is represented by the cement plastered over the boards, filling the crevices and uneven places in the boards, and receiving the tile on its surface. The endothelial cells are represented by the tile in the floor. The fibrils and cement substance, like the boards and cement, are both required to carry the overlying cells. It is evident, therefore, that the cement substance and the fibrils are required to make up a membrane in its physiologic sense, and must be thought of as coëxistent when the basement membrane is spoken of.

The cement substance can be removed from the fibrous tissue by means of pancreatin; and when stained with acid dyes there remains a layer composed of connective-tissue fibrils freed from the cement substance. In repeating Mall's experiments on the kidney, it seemed to me possible that the connective-tissue layer above noted corresponded to his reticulum, and that the cement substance

above noted represented his basement membrane. It seems to me best to regard the basement membrane as composed of a connective-tissue layer upon which a homogeneous layer lies in which the cellular layer is imbedded. This cement substance is negative to alkalies and to elastic-tissue stains, but has the property of precipitating more silver nitrate from a solution of given strength than any other tissue in the body. It is negative to Mallory's connective-tissue stain.

The problem of the cement layer is probably one of colloid chemistry, both as to its structure and function. In this layer and between the cells is a thick substance which has for its purpose the agglutination of the cells to the basement membrane, and perhaps of the cells to each other. Waldeyer has called this the "Kittsubstanz."

The importance of a clear comprehension of these two component substances appears in the study of the earliest stages of inflammation. What appears normally as a single layer of fibers, under stimulation of the reactive process, separates into fibrillæ so that a network replaces the single layer. The cement substance, increased by the influence of the reactive processes, exudes between the cells, and presents the first steps of the fibrinous exudate.

In addition to the white fibrous-tissue layer just noted, is a network of fibrils responsive to Weigert's elastic tissue stain. This network lies just below the fibrous tissue layer, and exhibits the physical and tinctorial properties common to this class of tissue.

The Subperitoneal Connective Tissue.—Beneath the membrana limitans, above described, is a layer of more or less loose connective tissue. Through this tissue courses a network of blood and lymph vessels.

The amount of connective tissue present in any section of the peritoneum is directly proportional to the mobility of the peritoneum in that situation. Over the tenæ of the colon, the inscriptiones tendeniæ of the rectus, and the central tendon of the diaphragm, as well as over the parenchymatous organs, such as the liver and spleen, this layer is very thin, being only sufficient to harbor a few vessels and to attach the membrana limitans to the fibers of the connective-tissue bands. In situations where the peritoneum is freely movable, the subperitoneal tissue is abundant.

The subperitoneal tissue is reticular in character, containing some fat cells in its lower layers in the region where it is most abundant.

In the mesentery the tissue between the two membrana limitans is a single mass harboring the vessels and nerves. Toldt calls this the membrana propria (Fig. 11).

The cellular elements in the subendothelial connective tissue are involved in a heart-breaking haze. Owing to the fact that the peritoneum has been the scene of conflict in the discussion on inflammation, endless literature has sprung up on the subject. There is a distinct variation in the cell content in lower animals.

The following is the result of my studies on the normal peritoneum.

Fig. 11.—Cross section of the mesentery. (Redrawn from Toldt.)

In the fibrous tissue layer of the basement layers are small ovoid cells containing an ovoid, deeply staining nucleus. These are the ordinary connective-tissue cells. They are more abundant about the small vessels, and sometimes exist in large numbers without evident cause for the variation in number. Sometimes these cells are spindle form or stellate. The latter forms are noted only in macerated, teased specimens. Less numerous are endothelioid cells which deserve this cognomen because of their close resemblance to endothelium. They have the same oblong nucleus and deeply staining nucleoli. The protoplasm, however, may show long prolongations. These vary much in number, being sometimes sparse, sometimes more abundant. Polynuclear leucocytes and plasma cells are sometimes noted, but it is questionable whether these are nat-

ural inhabitants of the normal extravascular peritoneal connective tissue. A more elaborate discussion of these cells will be necessary in the chapter on inflammation.

The Blood Vessels of the Peritoneum.—The vessels more or less related to the peritoneum are manifold. In some situations the peritoneum bears no other relation to the vessels than that of a covering membrane. This holds true of the great mesenteric vessels near their source. In some situations the peritoneum forms a veritable mesentery for the vessels. These vessels, as they approach their destination, come to assume a more intimate relation to the peritoneum, but it is only near their termination that they come in actual contact with the peritoneum. For the most part the visible vessels associated with the peritoneum are destined to supply the organ which the peritoneum covers, only the very minor part being destined for the membrane itself.

The peritoneum, as relates to its vascular supply, is typified by the mesentery. Here vessels visible to the eye divide the membrane into fields. As the intestinal border is approached the vessels become smaller, and the fields they enclose become smaller until finally the vessels reach their destination, the intestinal wall. Between these vascular loops the mesentery is perfectly transparent in the normal state. These are the familiar vessels of descriptive anatomy.

The apparently vascular areas outlined by the vessels above described are traversed by channels, not ordinarily participating in the transmission of blood corpuscles, but which under certain conditions may become active, therefore the blood vessels of the peritoneum may be divided into two classes: (1) those normally visible to the naked eye, which because constantly functionating may be called the service vessels; (2) those which do not ordinarily convey blood, but become functionating only under extraordinary conditions, which, therefore, may be called the potential vessels.

The Service Vessels.—These are the vessels familiar to the student of gross anatomy. They are lined with flat, oblong endothelial cells common to blood vessels (Fig. 12). They course along under the peritoneum, and, when properly injected, admit of dissection from the peritoneum. In the mesentery they are included in the membrana propria (Fig. 11), which receives reinforcement

of connective tissue and fat, or cells capable of taking on fat, in the region occupied by the vessels. They are accompanied by the lymph vessels, lacteals, and nerves. As they approach the intestinal border they intercommunicate, forming a meshwork gradually decreasing in size, and as they approach the gut they break into a series of vessels which encircle it, as above noted. The destination of these vessels, be it remembered, is the gut; and, properly speaking, they belong to the gut. The relation of the mesenteric vessels to the peritoneum is somewhat like the deep

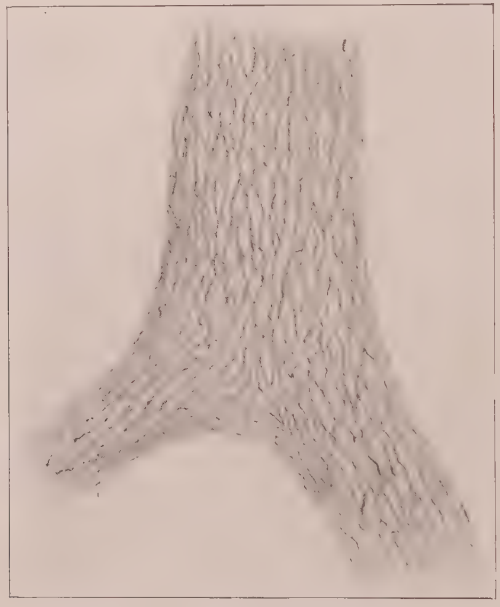

Fig. 12.—Endothelium of the service vessel of the mesentery.

vessels of the arm to the skin of this extremity. The vessels of interest are but the terminal branches of the deeper vessels. Inasmuch as a knowledge of the source of the terminals is dependent upon a knowledge of the trunk vessels, a brief study of the gross circulation is necessary. This, however, can best be considered in the discussion of the gross anatomy.

The Potential Vessels.—The actual vessels of the peritoneum, as exemplified by the vessel-free portion of the mesentery, are channels which, under normal conditions, do not transmit blood, but

do so under states of stress. Under the action of irritants, these vessels suddenly become active avenues of circulating blood. These are the only true terminal vessels of the peritoneum. What the state of function of these vessels is during periods of quiescence can only be conjectured. It is a fair speculation to assume that a circulation of serum, free from red blood corpuscles, takes place, and serves to nourish the several structures that go to make up the peritoneum. That white cells creep along these channels may be demonstrated with a considerable degree of certainty.

In order to demonstrate these vessels when in the quiescent

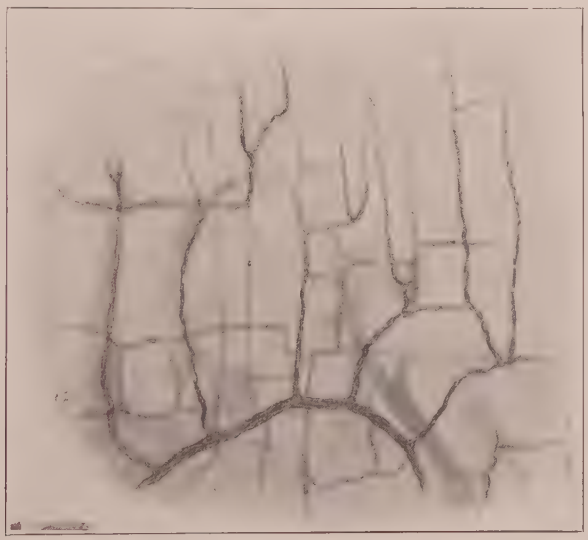

Fig. 13.—Potential vessels as they appear when an avascular area of the mesentery is injected with a solution of nitrate of silver.

state, it is only necessary to bleed an animal, and then inject the mesenteric vessels with a solution of silver nitrate. If this injection is made fairly tensely, the previously avascular areas become traversed by a fine network of vessels (Fig. 13) of something less than the diameter of a red blood corpuscle. These vessels, made visible with a low-power lens, divide the previously avascular area into quadrilateral fields of approximately equal areas. These vessels spring at approximately right angles from vessels visible to the naked eye. In some areas the plexiform arrangement of the

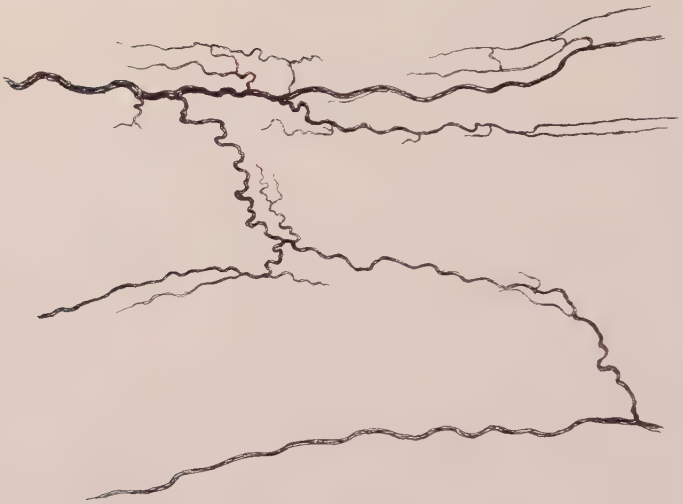

Fig. 14.—Higher magnification of the preceding figure. The free termination of the vessel is well marked.

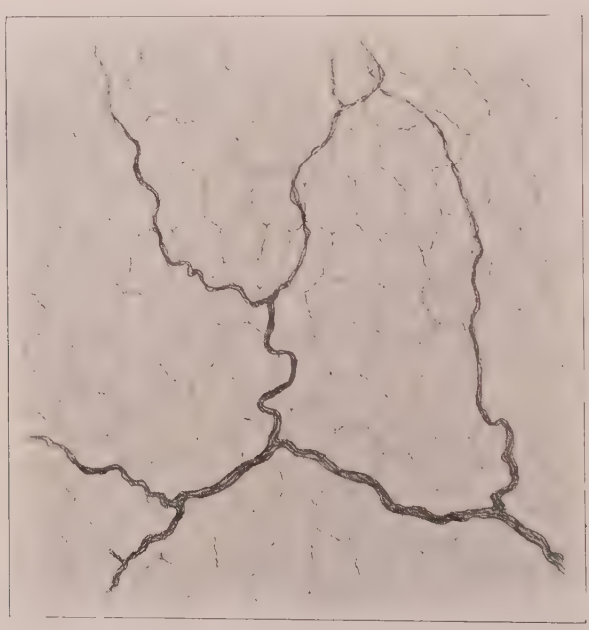

Fig. 15.—Potential vessels and endothelial cells making their comparative diameters apparent.

vessels is not complete. On the contrary, vessels seem to terminate free in the tissues by long cellular processes (Fig. 14). Such free dissemination of the vessels is probably only apparent, because of incomplete injections. At any rate, with increasing experience in making the injections, such vessels become fewer, and large areas are readily secured in which they do not occur at all; therefore, it is fair to assume that their occurrence is due to an imperfect technic.

These vessels are separated from the endothelial cells by the basement membrane only. Their small size is best appreciated by comparing them with the diameter of the endothelial cells which lie above them (Fig. 15).

The cell-lining of these small vessels differs from that of the service vessels; while the endothelial cells of service vessels resem-

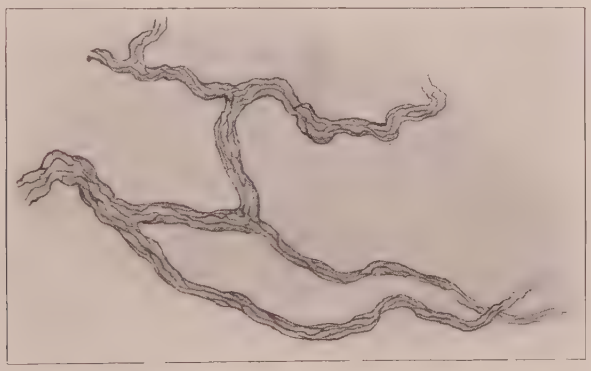

Fig. 16.—Endothelium of potential vessels.

ble closely those of the peritoneal endothelium, save that they are not as large and are more elongated and spindle form (Fig. 12). The cells lining the potential vessels are very much elongated, and sometimes extend along the lumen of the vessels for long distances or may be wound spirally about them. In some of these it is not possible to demonstrate nuclei, which is likely due to the difficulty of tracing cells their entire extent (Figs. 16 and 18).

The peritoneum of the parenchymatous organs is similarly supplied. If the peritoneum is removed from the surface of parenchymatous organs, particularly the gut, a fine plexus can usually be demonstrated (Fig. 17). The character of the plexus is less regu-

lar, and its constancy is dependent on the intimacy of the peritoneum to the underlying organ. Over the gut it is easily demonstrated, over the liver with greater difficulty, and over the tendinous portions of the diaphragm not at all. Interest in these vessels is largely centered in their pathologic relations, and will be more fully discussed in the chapter on general pathology.

Klein and Smith noted what I have called the potential vessels, but they regarded them as developing vessels, because they ended in long protoplasmic processes. These processes represent

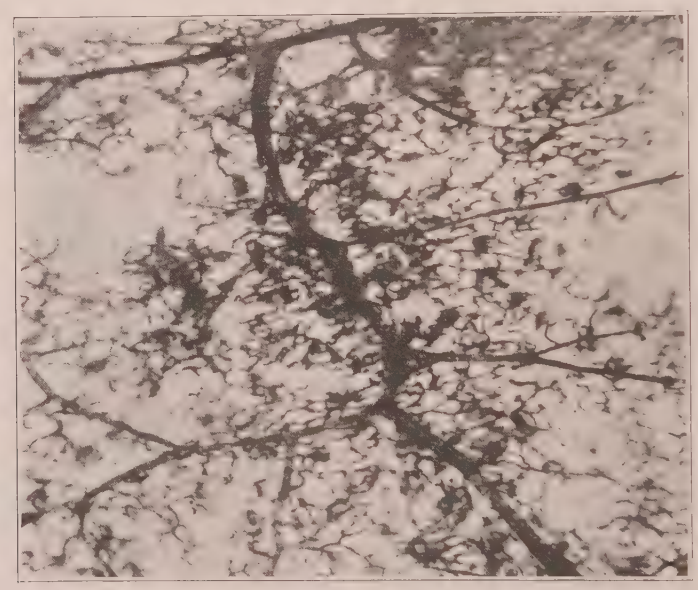

Fig. 17.—Photomicrograph of the blood vessels of the gall bladder.

the long cells terminating free in the tissues, above noted. The appearance of these end vessels is due to incomplete injection, as already noted, which may be caused by using too little pressure or too strong a solution of silver nitrate. With experience in technic these end vessels cease to occur in the normal peritoneum.

The veins of the peritoneum represent the counterpart of the arteries. The potential vessels above noted terminate in the veins. The endothelial cells lining the veins are broader and shorter than those lining the arteries. By these factors alone the experienced

eye can distinguish between the arteries and veins. This distinction holds good only when both vessels are stained, and fixed in their normal state of contractility. If an artery is tensely dilated, fixed and stained, the cells are as broad, relative to their length, as are those of the veins.

The Lymphatics of the Peritoneum.—The lymph vessels of the peritoneum form a network of channels beneath the basement membrane in the same stratum that the blood vessels occupy, or more deeply, as von Recklinghausen believes. They form a meshwork

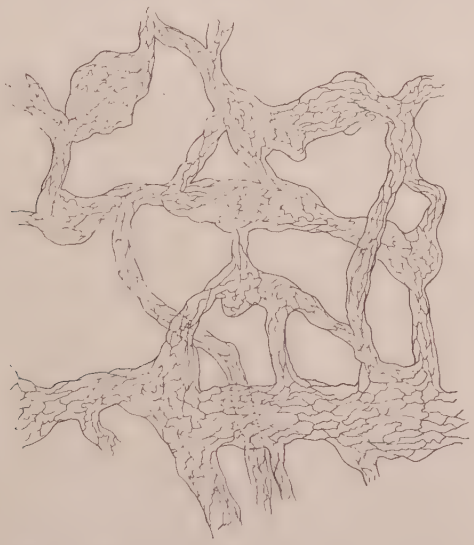

Fig. 18.—Lymphatic plexus of the mesentery. Nitrate of silver injection.

of relatively large vessels, which are irregular in their diameter. The course of the vessels is often broken by varicosities and dilatations of varying degree (Fig. 18). They are lined by flat cells with very irregular outlines, which are thinner than those of the peritoneal endothelium. The nuclei are flatter and ovoid in outline, and the nucleoli are less conspicuous. The lymphatics of the visceral peritoneum in the intestinal tract unite with those of the mesentery. Those over the solid parenchymatous organs unite with the lymph system of the organs, while those of the parietal peritoneum become a part of the lymph system of that region.

The lymphatics of the diaphragm may best be studied by direct injection by means of fine glass cannulas advised by Gerota, using the solution advised by Polano. With care the lymph channels can be demonstrated as a rich plexus. Sappey noted that the pleural lymphatics were more readily injected than those of the abdominal side. This is due, it seems to me, to the fact that the serosa is less closely adherent on this side. By the use of weak solutions of silver nitrate injected through a Gerota cannula the vessel outline may be demonstrated very clearly. By overstaining of the entire diaphragm in silver the lymph-plexus may be demonstrated. The lymph vessels then appear as lighter avenues in the darker background of the connective tissue.

Elaborate descriptions have been written of the connection between the lymphatics and the blood vessels. Von Recklinghausen has given the most complete description of such a connection. He described a lymph-canalicular system opening into the lymph vessels and extending to the blood vessels. These channels extended between the connective-tissue spaces, the cells of which formed a lining for those spaces. According to him, there was a flow of lymph-bearing nutriment from the blood vessels to the lymphatics. According to the conception of von Recklinghausen the lymph-channels have their origin in the free connective-tissue spaces, not as cell-lined spaces, but as clefts between the cells. He based his conclusions on the fact that when lymph channels are injected with a silver solution, not only are the intercellular lines defined, but, going out from these and extending into the connective tissue are fine lines of silver precipitate. It was these that he regarded as canals. The only other technic that suggested a similar result was that of Fohmann and Mascagni, who employed metallic mercury. They were able to cause this metal to pass into the connective-tissue clefts. Ranvier disposes of the discussion of von Recklinghausen's canal system by the simple statement that they do not exist, and expresses the belief that the lymph-system begins as walled off spaces.

In addition to von Recklinghausen's theory, two others were advanced. Based on the cellular studies of Virchow and Donders, Kölliker conceived a system of canals lined with endothelium extending from the blood vessels to the lymphatics. Brücke and

Ludwig believed that the lymphatics took their origin in open spaces in the connective tissue. Klein and Smith also expressed the belief in the continuity of the lymph wall and the connective tissue corpuscles.

The experiments of these investigators are easily understood. A silver solution is readily forced through a serous membrane. If any tissue remains long in contact with silver, all intercellular fluids will be affected, more or less and a precipitate formed. Later even the solid tissue will become impregnated with the silver. The extent of impregnation depends upon the duration of action and the strength of the solution. The results are affected by the state of preservation of the tissue. If processes of decomposition set in, the results are materially modified.

More recent technic has contributed much to the problem of the arrangement of the lymph vessels, and their relation to each other and to the surrounding tissue. By these means it may be shown that the large lymph channels are connected with each other by fine canals. It has also been shown that these connect with other channels, but there is no organic connection with the blood vessels, nor with the connective-tissue spaces.

That the lymph vessels are closed channels seems now determined with as much certainty as obtained in case of the blood-vessels. Interchange of both these systems with the intervening connective-tissue spaces is a chemical one, and has nothing to do with special spaces.

The lymph vessels of the peritoneum are distributed everywhere, there being a detailed variation only in the arrangement and in the ease with which they are demonstrated. Much confusion has arisen because writers have studied limited areas, and published these results. This has given rise to the belief that such structure exists alone in these regions. The diaphragm, notably, has been extensively studied, because of the ease with which the lymphatic system is demonstrated in this structure. This ease is readily explained by the topographic relation of the diaphragm, and by the fact that it possesses two peritoneal surfaces. There is no evidence to show that the diaphragm is possessed of more abundant lymphatics than other peritoneal surfaces.

Because the diaphragm is such a convenient object for the

study of lymphatics the chief controversy has centered about this organ. The earlier studies were made with the aid of silver-nitrate solutions. The result of this study was the hypothication of results already recorded.

With the advent of a special injection technic the discussion took a new angle. Special openings between the peritoneal surface and the lymphatics were no longer considered. On the contrary the question of the relation of the subserous plexus of the pleural and diaphragmatic serosa was discussed. Sappey, the pastmaster in lymph-channel injection, noted a general direction of the lympathics from the abdominal toward the pleural surface. Teichmann likewise expressed the possibility of a connection between the lymph system and the two surfaces, though expressing his inability to demonstrate positively their existence.

A more recent study is that of Küttner who was able to demonstrate connection between the abdominal serosa of the diaphragm and the thoracic lymphatics. My own studies lead me to the same conclusion with some reserve. Physiologic evidence points in the same direction. Dyes placed in the peritoneal cavity reach the mediastinal lymphatics.

To speak of such a connection as a "perforation" of the diaphragm by the lymphatics has led to misconception. There is no actual perforation of the diaphragm, any more than the various arterial plexuses of the intestinal wall perforate the intestine. The result of this misunderstanding has been to draw clinical consideration into the discussion.

Burckhardt suggests the rarity of a direct extension of inflammatory process from one diaphragmatic surface to the other as evidence of the nonresistance of a direct communication. Küttner, on the other hand, argues for the existence of such a connection by collecting instances in which lesions on each side of the diaphragm were sequential.

Whether or not an inflammatory affection involving one surface is likely to involve the other surface, depends entirely on the nature of the pathologic process, just as its extension across any other tissue barrier would, considering always the thinness of the diaphragm. There are always two serous layers to be traversed before a diaphragmatic peritonitis can become a diaphragmatic

pleuritis. If there is a lymphatic transportation from the abdominal diaphragmatic peritoneum, it leads in the mediastinal lymph glands, and not on the pleural surface.

The clinical aspect of this problem will be considered in connection with the complications of peritonitis.

Bibliography

ADAMI: Inflammation, London, Macmillan Co., 1907.

AFONASSIEW: Ueber den Anfang der Lymphgefässe in den serösen Häuten, Virchows Arch. f. path. Anat., 1868, xliv, 37.

ARNOLD: Ueber Diapedesis, Virchows Arch. f. path. Anat., 1873, lviii, 203.

Ueber die Kittsubstanz der Epithelien, Virchows Arch. f. path. Anat., 1875, lxiv, 203.

Ueber die Durchtrittsstellen der Wanderzellen durch entzüdete seröse Häute, Virchows Arch. f. path. Anat., 1878, lxxiv, 245.

AUERBACH: Zur Anatomie der Lymphgefässe im Besonderen derjenigen des Darmes, Virchows Arch. f. path. Anat., 1865, xxxiii, 344.

VAN BENEDEN: Contribution à la connaisance de l'ovaire des mammiferes, l'ovaire du vespertilio murinus et du rhinolophus ferrum-equinum, Arch. de Biol., 1880, i, 475.

BIZZOZERO: Ueber die innere Grenzschicht der menschlichen serösen Häute, Centralbl. f. d. med. Wissensch., 1874, xii, 210.

BIZZOZERO AND SALVIOLI: Sulla struttura delle membrane sierose e particolarmente del peritoneo diafframmatico, Gior. d. r. Accad. di med. di Torino, 1876, 3 s. xix, 466.

BRÜCKE: Ueber die Chylusgefässe und die Resorption der Chylus, Wien, 1853.

v. BRUNN: Zur Histologie der Epithelien der serösen Haute, Centralbl. f. allg. Path. u. path. Anat., 1900, xi, 604.

Ueber die Entzündung der serösen Häute mit besonderer Berücksichtigung der Rolle der Serosa Deckzellen, Beitr. z. path. Anat., 1901, xxx, 417.

BURCKHARDT: Ueber Kontinuitätsinfektion durch das Zwerchfell bei entzündlichen Prozessen der Pleura, Beitr. z. klin. Chir., 1901, xxx, 731.

BUXTON AND TORREY: Studies in Absorption, Jour. Med. Research, 1906, xv, 3.

COCCIUS: Ueber die Ernährungsweise der Hornhaut und die Serum fürender Gefässe im menschlichen Körper, Müller, Leipzig, 1852.

COHNHEIM: Ueber Entzündung und Eiterung, Virchows Arch. f. path. Anat., 1867, xl, 1.

DEKHUYZEN: Ueber das Endothel nach Untersuchungen, welche mittelst mordificierter Sellermethoden angestellt sind, Verhandl. d. X. internat. med. Cong., 1890, Berl., 1891, ii, 1, Abth. 4.

DYBKOWSKY: Ueber Aufsaugung und Absonderung der Pleurawand, Arb. a. d. physiol., Anst. z. Leipz. (1866), 1867, i, 40.

v. EBNER: Kölliker's Handbuch der Geweblehre des Menschen, Engelmann, Leipz., ed. 6, iii, 139.

EICHWALD: Ueber das Mucin besonders der Weinbergschnecke, Ann. d. chem. u. Pharm., 1865, cxxxiv, 177.

FLEMMING AND MERK: Sitzungsb. d. k. Akad. d. Wiss. in Wien Math. Wiss., Klasse, 1893, cii, Abt. iii; 1899, cviii, Abt. iii.

FLINZER: De argenti nitrici usu et effectu Praesertim in oculorum morbis sanandis, Lipsiae Vollrath, 1854.

Foà: Sul rapporto delle cavità plasmatische del tessuto connettivo coi vasi sanguignie l'infatici, Riv. clin, di Bologna, 1875, 2. s., v. 289. Trans., Virchows Arch. f. path. Anat., 1875, lxvi, 284.

Fohmann: Mémoires sur les communications des vaisseaux lymphatiques avec les veines, et sur les vaisseaux absorbans du placenta et du cardon ombilical, Liége, Desoer, 1832.

Gegenbaur: Lehrbuch der Anatomie des Menschen, Leipzig, Engelmann, ed. 4, 1890, p. 197.

Gerota: Ueber eine Verbesserung des Quecksilberinjektionsapparates für Lymphgefässe, Anat. Anz., 1896, xii, 35.

Hammar: Ist die Verbindung zwischen den Blastomeren wirklich proloplasmatisch und primär?, Arch. f. mikr. Anat., 1899, lv, 313.

Hensen: Ueber die Genese einiger Bindegewebssubstanzen, Anat. Anz., 1899, xvi, 417.

Hertzler: The Morphogenesis of the Stigmata and Stomata Occurring in Peritoneal and Vascular Endothelium, Tr. Am. Micr. Soc., 1901, xxii, 63.

His: Die Häute und Höhlen des Körpers, Basel, Schweighauser, 1865, p. 18.

Beiträge zur normalen und pathologischen Histologie der Cornea, Schweighauser, Basel, 1856; also Virchows Arch. f. path. Anat., 1863, xxviii, 361.

His: Ueber der Epithel des Lymphgefässwurzeln über die v. Recklinghausen-'schen Saftkanälchen, Ztschr. f. wissnschr. Zool., 1863, xiii, 455.

Johansson: Die Ringbänder der Nervenfaser, Arch. f. Anat. u. Physiol., Suppl. Band. 1892, p. 41.

Klein: Grundzüge der Histologie, Leipzig, Arnold, 1890.

Anatomy of the Lymphatic System, I, The Serous Membranes, London, Smith, Elder & Co., 1873.

Klein and Smith: Atlas of Histology, London, Smith, Elder & Co., 1880.

Koch: Ueber die Marksegmente der doppeltcontourirten Nervenfasern und deren Kittsubstanz, Jacob, Erlangen, 1879.

Kölliker: Handbuch der Gewebelehre des Menschen, Leipzig, Engelmann, ed. 5, 1867, p. 599.

Kolossow: Ueber die Struktur des Pleuroperitoneal und Gefässepithels (Endothels), Arch. f. Mikr. Anat., 1893, xlii, 318.

Küttner: Die perforierenden Lymphgefässe des Zwerchfells und ihre pathologischen Bedeutung, Beitr. z. klin. Chir., 1903, xl, 136.

Lavdowsky: Die Auswanderung der Leucocyten und die Frage nach dem Schicksal derselben im Organismus, Virchows Arch. f. path. Anat., 1884, xcvii, 177.

v. Lenhossék: Das Mikrocentrum der glatten Muskelzellen, Anat. Anz., 1899, xvi, 334.

Ludwig: Ber. d. Kön. Söchs. Gesellsch., Leipz., 1866-1867.

Ludwig and Schweigger-Seidel: Ueber das Centrum tendineum des Zwerchfelles, Arb. a. d. physiol. Anst. zu Leipzig, 1866-1867, i, 174.

Mall: Note of the Basement Membranes of the Tubules of the Kidney, Bull. Johns Hopkins Hosp., 1901, xii, 133.

Marchand: Ueber die Beziehung der pathologischen Anatomie zur Entwickelungsgeschichte, besonders der Keimblättlehre, Verhandl. d. deutsch. Gesellsch. f. Chir., 1899, ii, 38, Reimer Berl., 1900.

Mascagni: Vasorum lymphaticorum corporis humani historia et ichnographia, Senis, Carli, 1787.

Meyer: The Epithelium of the Peritoneal Cavity of the Cat; Contrib. from the Anatomical Lab. of University of Wisconsin, Bulletin of the University, 1900, No. 23.

Minot: The Mesoderm and the Coelom of Vertebrates, Am. Naturalist, 1890, xxiv, 877.

MUSCATELLO: Ueber die Bau und das Aufsaugungsvermögen des Peritonäum, Virchows Arch. f. path. Anat., 1895, cxlii, 327.

NEUMANN: Die Beziehungen des Flimmerepithels der Bauchhöhle zum Eileiterepithel beim Frosche, Arch. f. mikr. Anat., 1875, xi, 354.

NICOLAS: Note sur la morphologie des Cellules endotheliales du peritoine intestinal, Compt. rend. Soc. de biol., 1895, iii, Ser. x, p. 196.

OEDMANSSON: Beiträge zur Lehre von dem Epithel, Virchows Arch. f. path. Anat., 1863, xxviii, 361.

PALADINO: Sur l'endothélium vibratile chez les mammifères et sur quelques faits physiologiques relatifs aux formations endothéliales, Arch. ital. de biol., 1883, iii, 43.

PICK: Mittheilung zur Pathologie des Beckenbauchfells, Berl. klin. Wchnschr., 1900, xxxv, 219.

POLANO: Zur Technick der Darstellung von Lymphbahnen, Deutsch. med. Wchnschr., 1902, xxviii, 482.

RABL-RUECKHARD: Einiges über Flimmerepithel und Becherzellen, Arch. f. anat. Physiol. u. wissensch. Med., 1868, 72.

RANVIER: Technisches Lehrbuch der Histologie, Leipzig, Vogel, 1888.

Des Clasmotocytes, J. de Microg., 1890, xiv, 103.

Traité technique d'histologie, Paris, Savy, 1889.

RAWITZ: Grundriss der Histologie, Berlin, Karger, 1894.

v. RECKLINGHAUSEN: Die Lymphgefässe und ihre Beziehung zum Bindegewebe, Berlin, Hirschwald, 1862.

Lymphgefässystem im Strickers Handbuch der Lehre von den Geweben, Leipzig, Engelmann, 1871, p. 214.

ROLLETT: Untersuchungen über die Structur des Bindegewebes [Sitzungsb. d. k. Österr. akad. d. Wissensch. xxx] Untersuch. z. Naturl. d. Mensch. u. d. Thiere, Giessen, 1860, vi, 1.

SAPPEY: Des vaisseaux lymphatiques, in: Traité d'anatomie, Paris, Delahaye, 1869, ii, 760.

SCHAFFER: Zur Kenntnis der glatten Muskelzellen insbesondere ihrer? Verbindung, Ztschr. f. wissensch. Zool., 1899, lxvi, 214.

SCHIEFFERDECKER: Beiträge zu Kenntniss des Baus der Nervenfasern, Arch. f. mikr. Anat., 1887, xxx, 435.

SCHWEIGGER-SEIDEL, AND DOGIEL: Ueber die Peritoncalhöhle bei Fröschen und ihren Zusammenhang mit dem Lymphgefässysteme, Arb. a. d. physiol. Anst. zu Leips., (1866), 1867, p. 68.

STÖHR: Text-book of Histology, Phila., P. Blakiston's Son, 1903.

SUBBOTIN: Internat. Klinik., St. Petersburg, 1882, No. 12.

TEICHMANN: Das Saugadersystem vom anatomischen Standpunkte, Leipzig, Engelmann, 1861.

TOLDT: Bau und Wachsthumsveränderungen der gekröse des menschlichen Darmkanales, Denkschr. d. Math-naturw, cl. d. k. Akad. d. wissensch., 1879, xlii.

TOURNEUX: Recherches sur l'epithélium des séreuses, J. de l'anat. et physiol., 1874, x, 66.

TOURNEUX AND HERRMANN: Recherches sur quelques épithéliums plats dans la série animal, Jour. de l'anat. et de le physiol., 1876, xii, 199-386.

USSOW: Zur Lehre von den Stomata der serösen Höhlen, Abstr., Ergebn. d. Anat. u. Entwick., 1899, ix, 651.

VIRCHOW: Gesammelte Abhandlungen wissenschaftlicher Medicine, Frankfurt v. Meidinger, 1856, p. 167.

WALDEYER: Die Kolonnischen die Arteriae colicae und die Arterienfelder der Bauchhöle nebst Bemerkungen zur Topographie des Duodenum und Pankreas, Berlin, Reimar, 1900.

Archiblasts und Parablast, Arch. f. mikr. Anat., 1883, xxii, 1.

CHAPTER III

DEVELOPMENT OF THE PERITONEUM

The topographic relations of the abdominal viscera and their associated mesenteries and omenta form a veritable puzzle to the student. It is conceded by teachers of anatomy that the only way to convey an adequate conception of the adult relations is by tracing the process of development.

To simplify the problem teachers of anatomy of the abdomen have resorted to diagrammatic illustrations. The value of diagrammatic drawings is dependent on the fact that only by their aid can the migration of the abdominal organs be presented as a sequential process. Diagrammatic drawings must, however, be accepted at their true worth. Students are too apt to regard these diagrams as representing facts and base their mental conceptions on them. The consequence is that when they enter the practice of surgery their ideas of the relations of the organs of the abdomen retain the earmarks of this early teaching from diagrams. Proof of this is obtained if one reads the literature on ptoses, adhesions and kinks.

In this monograph, therefore, an endeavor will be made to supplement the diagrammatic drawings with various observations on human material and only those problems that concern the thought and practice of surgeons will be considered.

General Considerations.—Up to about the third or fourth week of development the abdomen of the embryo consists of a cavity across which is stretched a mass of cells (Fig. 19), extending from the aorta to the anterior abdominal wall, in the center of which is the enteric tube. The cavity on either side of this septum is the pleuroperitoneal cavity. This septum is composed of mesenchymal cells and the cavities also are lined by mesenchymal cells, the future endothelium.

Very early that portion of the septum anterior to the endodermic tube below the future stomach is lost (Fig. 20). That part remaining over the site of the stomach is permanent and becomes

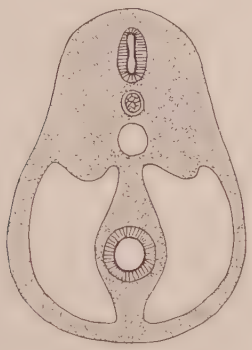

Fig. 19.—Schematic drawing representing the mesentery, and the primitive gut tract. (Minot.)

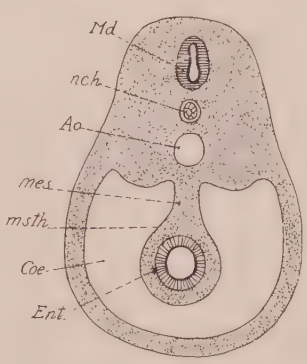

Fig. 20.—Same as Fig. 19 below the future stomach. (Minot.)

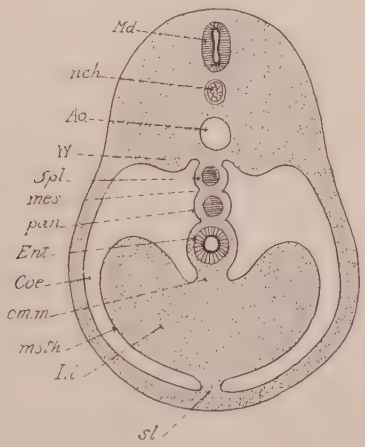

Fig. 21.—Same as the preceding, but showing the liver, *li*; the spleen, *spl*; and the pancreas, *pan*. (Minot.)

the lesser omentum. The septum below the stomach disappears and a single abdominal cavity results.

The future liver buds off from the enteric tube and develops into this septum and secures its peritoneal coat and capsule from it.

In that portion of the septum posterior to the enteric tube the spleen and pancreas appear (Fig. 21).

Following this stage, at about six weeks, the stomach begins to assume its permanent form, appearing as a posterior bulging of the endoderm. Coincident with this the segment of the entoderm below the stomach elongates and pulls with it the primitive septum. It is from this that the gut and its mesentery is developed. At

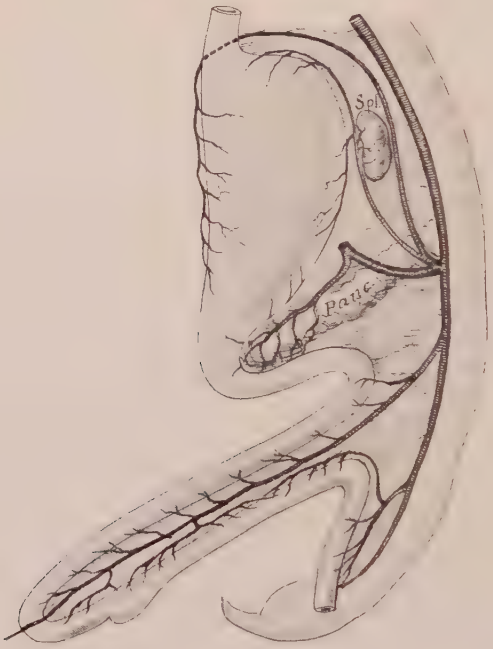

Fig. 22.—The primitive digestive organs and their vessels. (Hertwig.)

this early stage the celiac axis and the mesenteric vessels are already apparent and indicate the permanent site of the mesenteric root (Fig. 22).

Following this stage the gradual elongation of the gut tract requires a rearrangement of parts in order to secure accommodations within the abdominal cavity. The stomach comes to lie more transverse. The pyloric end crosses slightly the midline of the body toward the right and the fundus extends toward the left. By the gradual lengthening of the gut, the lower segment of the

future stomach is reached, and then crosses directly toward the right side of the abdomen below that organ (Fig. 23). At a point below the pyloric end of the stomach a budding of the gut appears, the future cecum. This portion of the future colon lies over that portion of the gut adjoining the right end of the stomach, the future duodenum. It is at this point that the colon secures its first attachment. While this transposition is taking place the primal mesentery becomes twisted upon itself. During this period the elongation of the small gut causes it to form coils, filling the arc

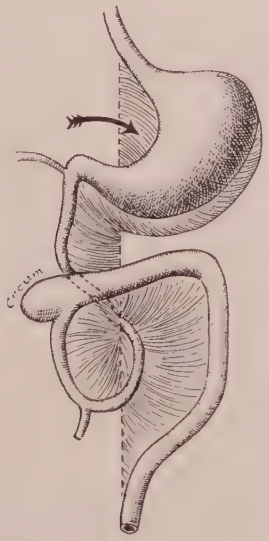

Fig. 23.—Beginning migration of the cecum. (Hertwig.)

formed by the position of the colon. The mesentery, of course, follows the gyrations of the small gut.

While these changes in the gut have been taking place, equally important changes have been taking place in the region of the stomach. As the greater curvature of the stomach enlarges it moves toward the left, carrying with it the primitive septum in which the pancreas and spleen are developing. This septum soon exceeds in extent the apparent needs and begins to pouch to the left and below. This is the future great omentum. By continuing to

increase in length it extends over the transverse colon and beyond, approaching the extent as we know it in the adult state.

Simultaneously that part of the septum, in which the pancreas and spleen are being developed, also suffers a dislocation by the lateral development of the stomach. The septum, instead of extending anteroposteriorly, extends from the attachment at the midline to the left. As a result the pancreas comes to lie against the posterior abdominal wall and the spleen against the left lateral wall and finally the layers of mesenchyme of the pancreas and the pos-

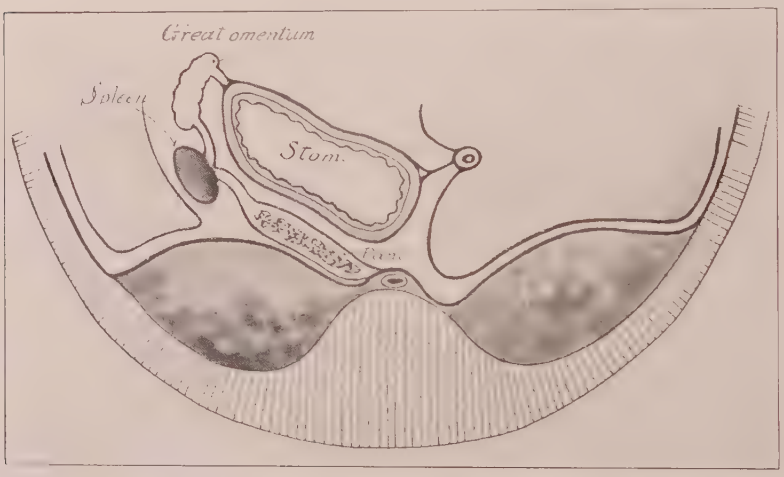

Fig. 24.—The pancreas and spleen are approaching their permanent position. (Toldt.)

terior wall become fused and the pancreas is no longer within the abdominal cavity, and is said to be retroperitoneal (Fig. 24).

The relation which the various organs have now assumed approximates that of the adult state. The stomach lies transversely, to its left is the spleen, and behind, the pancreas. At its right is the attachment with the liver. What remains of the anterior septum represents the hepatogastric ligament and the lesser omentum.

It remains now but to trace the subsequent course of the great omentum, which, it will be remembered, protrudes beneath the stomach. This is reproduced in cross section in Fig. 25. In these figures, *b* represents the lowest point of the omental loop.

As it extends over the transverse colon (*c*, Fig. 26) it impinges more and more upon the gut. It begins to unite with the mesentery of this gut, and finally with the gut itself, so that the upper part of the posterior layer of the great omentum becomes a part of the mesocolon. At this time the anterior layer of the great omentum still hangs as a loop so that there is a free space extend-

Fig. 25.—Diagrammatic section of the stomach showing the successive steps in the development of the great omentum.

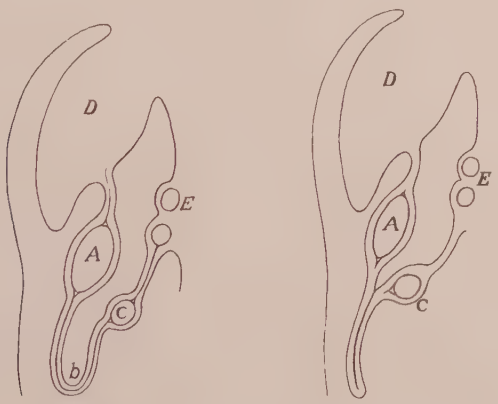

Fig. 26.—Same as Fig. 25, showing the obliteration of the omental sac.

ing from behind the stomach to the bottom of the omental fold. Soon after birth, however, the anterior and posterior layers become fused as far up as the transverse colon (Fig. 26). That portion between the stomach and transverse colon remains as the gastrocolic omentum and forms the lower limit of the lesser peritoneal cavity.

In this simple manner the complex relations, as they are ob-

served in the adult, are brought about. The further evolution from this point involves many factors of moment to the surgeon. Some of these are but little understood and one can do little more than indicate them. They comprise problems in histogenesis the details of which are but little known. The topographic relations, however, may be followed with some confidence and these are the factors which concern the surgeon.

Special Problems in Splanchnogenesis.—The most fundamental of the problems in the final relation of the viscera to the peritoneum and the abdominal wall has to do with the behavior of the mesenteries. Two general notions have prevailed. The older authors believed that when an organ had a mesentery and subsequently lost it and became wholly or in part retroperitoneal, it became so because the two layers, of which the mesentery was believed to be composed, spread out and allowed the organ to divest itself in part of its serous covering (Fig. 27).

Toldt has shown that as a general law governing the development of the peritoneum this conception is wrong. He showed that what actually occurs is a fusion of one surface of the mesentery with the parietal wall. Toldt's idea is illustrated in Fig. 28. At *A* in this figure the gut with its mesentery is again represented as in *A* in Fig. 27. *B*, Fig. 28 represents the gut in the act of fusion. The gut comes in contact with one surface so that layer *b* comes in contact with the posterior wall. Layer *b* then fuses with the wall as far as it is in contact, and the gut becomes retroperitoneal for that extent over which fusion has taken place, (*C*, Fig. 28).

While Toldt's ideas, as above set forth, are correct in the main, I desire to call attention to two fallacies which have developed because of a too general acceptance of his theory.

In the first place it is a mistake to speak of the fusion of layers of peritoneum in this early period of development. The truth is that what represents the mesentery is but a mesenchymal mass. Fusion occurs between mesenchymal tissues before they have developed into the differentiated layers of tissue, which conjunctively we speak of as the mesentery. At the time of fusion these tissues are covered by a layer of embryonal tissue resembling only slightly adult peritoneum. The power of such tissue to fuse with

like surfaces is much greater than in fully formed tissue. Moreover, it is also worthy of note that two layers, which older anatomists conceived as separating, do not exist in fact as definitely formed membranes.

The second fallacy is to ignore the possibility that sliding of the peritoneum, as described by the older authors, does not take place at all. The schematic drawings, representing organs with long mesenteries making great excursions, give a wrong impression. What actually occurs is that those surfaces, which have become fused with other surfaces, do so at that early period before the organs assume the position normal to the adult, and having

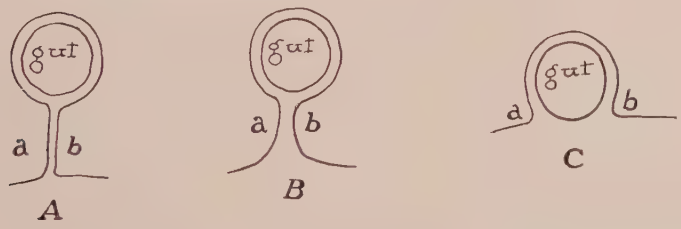

Fig. 27.—The mesentery becomes obliterated by the gradual separation of the layers of the mesentery. (Toldt.)

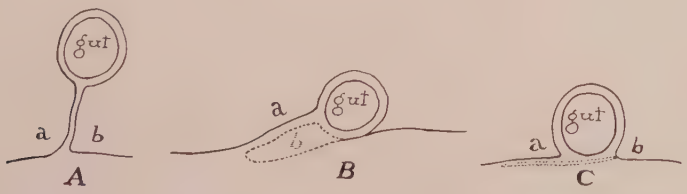

Fig. 28.—The gut looses its mesentery by fusion with the parietal wall. (Toldt.)

fused may slide for considerable distances. This is best illustrated in case of the cecum. This organ becomes fused with the posterior wall when it lies just lateral to the vertebral column over the site of the future pylorus. As the colon develops it forces the cecum to descend over the kidney. In making this descent it slides partly over the (anlage of) peritoneum and partly carries this structure with it.

In all the processes above described the tissues which form the peritoneum are still in such an early state of development that it

represents little more than an anlage. At any rate the use of this word serves to emphasize the incomplete state of its development.

The capacity of the more fully developed peritoneum to fuse must be recognized, however. This is noted particularly in the fusion of the sigmoid mesentery about the seventh month and in the fusion of the two layers of the great omentum some time after birth. Even in these the problem is different from fusion in the fully developed peritoneum. While the morphology of the sigmoid mesentery of the seven month fetus resembles that of the adult, the histochemistry of the former resembles more that of embryonal tissue than it does that of a fully developed peritoneum. The same may be said of the fusion of the omental layers after birth. The omentum, as a matter of fact, retains throughout life a plasticity and capacity for fusion not shared by other peritoneal surfaces. It is manifestly incorrect to attempt to find an identity between fetal fusion and the formation of adhesions after birth.

Development of Special Regions.—With the foregoing outline in mind the development of special regions may be considered. It is in this study that problems of interest to the surgeon are found.

The Omentum.—The development of the omentum may well be considered first because this part has been most extensively studied, and because the changes it undergoes are fundamental and the principles here learned may be applied to other parts of the peritoneum.

The modern discussion of the great omentum may be said to begin with Meckel. He first described the rotation of the colon and noted that the omentum appears as a distinct projection from the curvature of the stomach at the second month but he misinterpreted its formation. He believed that the posterior omental layer is derived from the pancreas. As the pancreas develops away from the stomach, the omentum, he believed, separated into two layers. After the rotation of the colon and its mesentery, the omentum, he correctly observed, unites with it by its lower layer. This adhesion, he noted, occurs from above downward. He was the first to note that obliteration of spaces between two opposed surfaces of the omentum may take place by agglutination. We find here the first reference to the obliteration of peritoneal

surfaces. He also noted that in many of the lower animals the omentum remained permanently as two layers.

Müller noted that the stomach of the embryo at the sixth week is possessed of a true mesentery. He very correctly remarks that this is "der Schlüssel zur Bildungsgeschichte des grossen Netzes." He believed that the mesentery begins at the median line of the posterior wall of the abdominal cavity, passes to the posterior border of the stomach, spreads apart to take in that organ, unites again at the posterior border, and proceeds to the liver. As the stomach assumes a more transverse direction, the mesogastrium changes its direction from the vertical to one diverging to the left and upwards. As the mesogastrium develops a pouching occurs which points downwards and to the left. As the omentum develops downward and the stomach comes to lie more transversely, a space is formed behind the stomach, the lesser peritoneal cavity. As the omentum descends and the transverse colon ascends the space in the parietal peritoneum between the insertion of the omentum and the insertion of the mesocolon becomes lessened. This approach continues until the space is obliterated. This takes place from right to left because the insertion of the mesogastrium is lower in the right side.

Müller's first opinion was that this obliteration took place by a stripping off of the parietal peritoneum and that by the regression of the fold the colon was finally reached. Later researches, however, convinced him of the correctness of Meckel's view, that the space was obliterated by the agglutination of two opposing surfaces.

Hansen denied that the posterior layer of the omentum was directly continuous with the mesocolon. Hennecke came to the same conclusions as Meckel. He emphasized the observation of Meckel that in the lower animals the permanent state is but a representation of a stage in development in the human fetus. He noted that in the ruminants the great omentum and transverse colon are permanently separated, also that in mammalia the mesogastrium formed a true bursa omentalis while in other vertebrates it remained as an ordinary mesentery. Huschke agreed with the preceding, as to the method of union of the omentum and mesocolon and advanced the theory that the cause of the union of the omen-

tum and the mesocolon was the high position and slight mobility of the colon. Bochdalek believed with Froriep that the great omentum includes the transverse colon and forms its meson. Bochdalek also noted that the ligament previously called the pleurocolic would be more correctly called the phrenocolic and that it was a part of the great omentum. He divided the omentum of the newborn into three parts, the left, or ligamentum phrenocolicum above noted, a right containing the omentum colicum Halleri, and a middle portion.

The foregoing represents the knowledge of the great omentum existing at the time Toldt wrote his epoch-making papers. By means of careful examination of a series of young embryos he was able to confirm the observations of Meckel and Müller and definitely establishes the relation of the omentum to the mesocolon. In addition, he demonstrated the relation of the developing spleen and pancreas to the mesogastrium. It is on his work that all subsequent studies have been based.

However, the earliest embryo studied with particular attention to the great omentum was that of Swaen. This author's researches are of particular interest because they were made on an embryo of 3.8 mm. He found the anlage of the bursa omentalis to be a blind pouch on the right side of the mesogastrium but pointing to the left. This sac continued cranialward as a groove on the dorsal wall bordered on the left by the dorsal mesentery and on the right by the plica venæ portæ. It is the caudal elongation of the sac with the rotation of the stomach and the elongation of the mesogastrium, that forms the great omentum.

Swaen's work emphasizes that when the spleen forms and begins to be displaced laterally the posterior mesogastrium is still represented by a mass of undifferentiated cells. As the lateral movement begins the space between the left side of the mesogastrium and the posterior wall becomes obliterated but the process resembles the pressing together of two colloidal masses more than the union of surfaces worthy the name peritoneum.

Toldt was concerned with the migration of the colon in a six weeks' fetus; he observed that the duodenojejunal angle was already fixed against the posterior wall, but that the duodenum looped free with a mesentery continuous with the mesogastrium.

There seems to me to be no evidence that the duodenojejunal angle ever was mobile. The fixity of this point seems to be associated with the development of the mesenteric root and the contained vessels. Within the substance of the mesogastrium are the rudiments of the pancreas and spleen. The head of the pancreas already fills the concavity of the duodenal loop while the body extends to the left entirely imbedded in the mesogastrium. The spleen is represented by a collection of cuboidal cells situated within its substance not far from its insertion into the great curvature. The relation of the pancreas and spleen to the mesogastrium is represented by a schematic drawing (Fig. 24).

The third month these relations are on the whole maintained. The greater curvature has increased in size and the omentum has more than kept pace with this development and projects before the curvature as a real bursa. The posterior wall contains the pancreas which produces a prominence in the membrane, the plica gastropancreatica, which forms a dividing line for the first time between the greater and lesser cavities. By this the great omentum is given a sort of constriction through which it communicates with the lesser peritoneal cavity. In this way the lesser peritoneal cavity obtains definite boundaries. The plica just mentioned defines the lower border, behind is the former mesogastrium, now about to become the parietal peritoneum; in front is the lesser omentum, the erstwhile anterior mesogastrium. At its extreme right border this membrane is thickened into the ligamentum hepatoduodenale which spans the interval between liver and gut, leaving an opening below, the foramen of Winslow. This lesser cavity contains a part of the liver and hence may be regarded as a part of the peritoneal cavity. The great omentum on the other hand projects downward as a loose sac, forming no part of the abdominal cavity but a part of its contents.

Important changes are now observed in the relations of the posterior mesogastrium and its contained organs and the abdominal wall. That portion which incloses the pancreas becomes attached to the posterior wall over the adrenal. It has obtained a lateral attachment in addition to the early attachment in the median line. But more important is the method of attachment. The older authors, notably Müller, believed that the two layers of the peri-

toneum separated and the pancreas escaped and became retroperitoneal. Toldt demonstrated by means of manipulation with a probe that the attachment takes place by the folding over of the mesogastrium and an agglutination of the two layers. He was able to separate the loosely attached surfaces until the midline was reached.

I have had abundant material for review of these observations of Toldt. The pancreas can be separated from the posterior parietal as he states. To assume that one membrane is being separated from another is overstating the facts. Neither can the opinion of Müller that the two layers of membranes separated and permitted the pancreas to become retroperitoneal be demonstrated. At this time the pancreas parenchyma as well as the anlage for the peritoneum are but slightly differentiated. The latter cells are overlaid by a fine fibrinoid pellicle, semifluid in character. This, coming in contact with a similar structure of the posterior abdominal wall, makes a cell mass devoid of any cell differentiation suggestive of the fully developed peritoneum.

There is still the tail of the pancreas and the spleen free in the mesogastrium and the head of the pancreas lies free in the mesentery of the movable duodenum. In the weeks following these parts become more closely adherent to the posterior wall. In addition, the great omentum increases in size particularly at its right border but it is still everywhere free from the transverse mesocolon.

In eleven-centimeter embryos the posterior layer of the omentum has become adherent to the anterior layer of the transverse mesocolon as far as the gut but is not adherent to the latter. This adhesion begins on the right side and proceeds to the left. Here there is a measure of justification in speaking of a fusion of serous surfaces for there are indications of an endothelial layer and a basement layer. It is worth noting, however, that while there is a well advanced morphologic differentiation, there is but little chemical relationship between this and the earlier stages, hence not yet a condition analogous to the fully developed peritoneum. The whole omentum has increased in size and covers the coils of the jejunum as far as or beyond the median line. The tail of the pancreas has become adherent to the posterior wall and the meso-

gastrium as far as the spleen. That portion leading from the fundus of the stomach to the spleen has become prominent and may be recognized as the ligamentum gastrolienale.

The chief changes in the fifth and sixth months occur in the part of the omentum over the duodenum. The omentum becomes attached to the transverse mesocolon over the duodenum and at the same time the duodenum becomes fused to the posterior wall. The omentum may become fused farther to the right side, as far even as the ascending colon and in some instances to the lateral parietal wall. When this is the case the right lateral portion has been called the ligamentum colicum Halleri. This extensive adhesion is sometimes extended to the gall bladder and to the surface of the liver itself.

At birth the two layers of the omentum are still free and can be inflated by inserting a blowpipe into the foramen of Winslow. The agglutination of these layers usually begins soon after birth. The process usually begins in the region of the hepatic flexure. The layers of the ligamentum colicum Halleri are first obliterated and are often adherent to the lateral wall as far as the cecum. The part in the region of the spleen next becomes agglutinated and finally the central portion.

The development of the omentum minus was correctly noted by Müller and Hennecke as a short septum extending between the liver and stomach. Toldt demonstrated in an embryo of the fourth week that this septum was continued upward with the posterior heart mesentery and he regarded the omentum minus, therefore, as a caudal extension of the posterior mesocardium.

The union of the great omentum and the mesocolon and the colon was believed by Toldt to occur by the adhesion of the omentum to the mesentery of the colon. Lockwood on the contrary believed that there was no adhesion of the two surfaces but that there was an unfolding of the posterior layer of the mesentery and the anterior layer of the mesocolon. According to this opinion the transverse mesocolon is formed of only two layers. He points out that because the stomach is nearly vertical and the colon is nearer the pyloric than the cardiac end of the stomach, it takes longer to become obliterated at the left side. Zoerner was of the same view. According to this view the colon would

come to lie, because of this process of unfolding, between the layers of the original dorsal mesentery, and the mesocolon is formed, therefore, by but two layers.

Toldt replying to Lockwood declares that the reason for the mistaken view is the old delusion that the mesentery is composed of two layers in which the viscera are developed. He brings further evidence by presenting a cross-section of the omentum and mesocolon which shows at certain points an incomplete union of the two membranes showing each a separate layer. This cut presents convincing evidence of the correctness of Toldt's views. The lower layer of the adult gastrocolic omentum differs from the upper in that the lower is smoother, shows the vessels plainer, and shows less deposition of fat. In comparative anatomy he finds the additional argument that the degree of adhesion of omentum and mesocolon presents a great variety of variations. Toldt in continuing his argument in favor of his view touched a point not made by any other observer. He noted that the mesentery is not composed of two layers but that it is a mass of tissue covered with a layer of endothelial cells from which later the various layers are formed. Furthermore that the union takes place gradually from above downward is shown by the fact that these layers can be separated by passing a probe between them. He noted, however, that the strength of this argument is largely subjective.

The cause of the union between the layers of the mesocolon and omentum is still controversial. Huschke believed that it was due to the high position of the transverse colon and its limited mobility. Treitz thought that the increased friction of a segment of gut against a peritoneal fold or surface might play a part in the secondary adhesions. Broesike thought this possibility might exist in adults but did not believe it to be of influence in embryos because there is no peristalsis and no variability in degree of distention due to the varying amount of fecal contents. He believed that the opaque appearance of the peritoneum at the point of adhesion indicates the presence of some form of formative action (as a keratitis). This formative action leads to a rapid development of connective tissue and to a transformation or casting off of the peritoneal covering. This irritative action,

in the light of recent research, it must be admitted, may come as the result of a rapid growth of the underlying organ.

Toldt believes the reason for agglutination of peritoneal surfaces is still unknown. He thinks Broesike has added nothing because there is no evidence of "increased growth" when examined microscopically.

My own studies have had less to do with the morphogenesis than with the histogenesis. Insofar as the former goes my studies have been confirmatory of the work of the investigators above quoted. Insofar as the histogenesis goes I do not believe enough attention has been paid to the character and form of the cell. According to Toldt the mesenchyme cells at four weeks are closely crowded together with little basal substance. I have had no human material at this age, but at six weeks there is considerable basal substance and the more deeply lying cells are spindle form and the surface cells are flat, the protoplasmic area being approximately as thick as the nuclear area. The thinning of the cells shows first at the cell borders and by the end of the eighth week the protoplasmic area has thinned and the nuclear area bulges as in the fully formed endothelium.

Beneath the young endothelium is a layer of undifferentiated cells which by the third or fourth month take the form of the basement membrane. While this layer takes on its permanent morphology it does not assume its permanent tinctorial character until later.

The deeper layers which surround the vessels and nerves are composed of loose fibrils in which lie spindle-form cells. The cells at this time still represent a common type. This tissue is characterized chiefly by the reaction to the specific dyes. While it takes Mallory's stain, it stains a reddish yellow with Van Gieson's stain.

The important part in these studies is that the full adult type has not been reached when the agglutinations occur. While the general morphology may approach closely the fully developed stage the chemical reaction has not attained a final stage. The histochemistry is identical with that seen in tissues in a state of reaction from inflammation. It is safe to say that once a peritoneal surface has fully developed, morphologically and chemically no further fusion takes place.

What factors are operative in determining the time of final chemical development is not known. This state is not reached until the underlying organ has attained full growth and development. Whether rapidly growing regions exert a chemotactic influence on the other surfaces is not known. The histochemistry

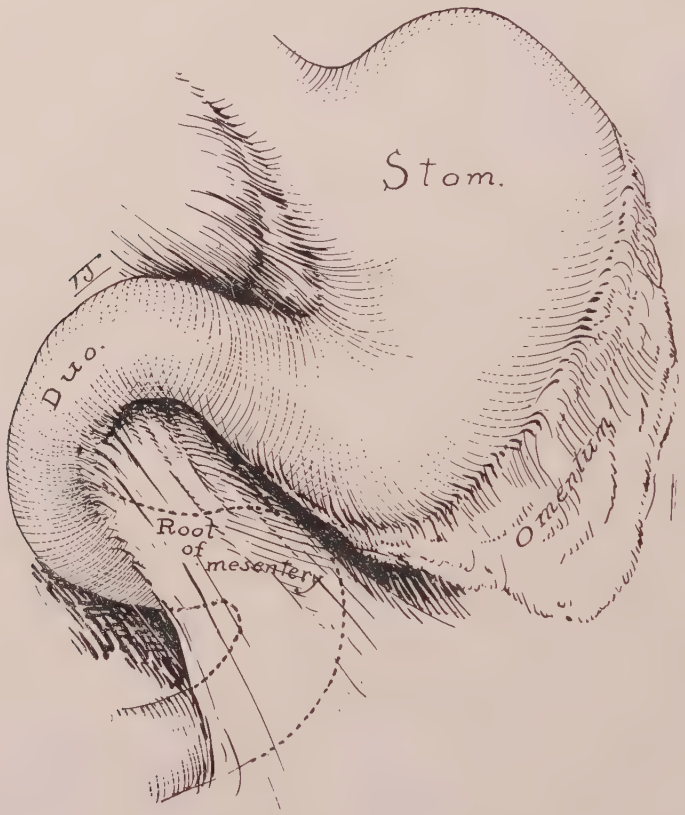

Fig. 29.—The duodenum projects upward and to the right free from attachment other than by its meson.

of the tissues makes such an hypothesis something less than ridiculous to say the least. This state, together with the pressure exerted by growing organs, may account for fusion in certain situations.

Development of the Hepatoduodenal Region.—The relationship of the structures in this region, as found in the adult is readily

understood if the fairly simple developmental evolution is followed.

The early stage is represented semidiagrammatically in Fig. 22. The most fixed point is represented by the pylorus and the duodenojejunal angle. These points never possess a true meson. As the duodenum develops it extends toward the right and upward toward the liver (Fig. 29). On account of its increase in length it must force itself within the small space between the liver and the posterior parietal wall. As it projects upward it secures a

Fig. 30.—The colon is attached to the pylorus by the duodenocolic ligament. The omentum has begun to fuse at the angle made by the fusion of the colon and duodenum.

true meson and projects up without attachment other than that furnished by its meson in the midline. I have noted this mobility of the duodenum in fetuses as long as 24 mm. Surgeons reproduce this stage when they loosen the duodenum and lay it forward when attacking the terminal portion of the bile duct. At the same time that the duodenal loop is acquiring this position the duodenojejunal angle becomes dislocated slightly to the left. The loop formed by the duodenum becomes occupied by the gradually

growing root of the common mesentery (Fig. 29). Later the duodenal loop becomes attached to the parietal peritoneum and the hepatoduodenal ligament is formed (Fig. 30). At this stage the transverse colon is entirely free from attachment in this region. This union soon takes place, however. The usual position of the cecal bud at the time of attachment is just to the right of the midline. The cecal bud at this time may point upward and outward or it may have already formed a curve and point downward toward

Fig. 31.—The cecum approached the pelvic brim before becoming attached. The wide fusion of the gut is well shown.

its future position (Fig. 30). Less commonly it extends far downward toward the right iliac fossa (Fig. 31). Even with such extensive migration the point of primary fusion is usually just below the pylorus. At this early stage the entire gut is enveloped in a plastic mass and appears to be retroperitoneal. It is only in its later development that this portion of the gut obtains a mesentery. Coincident with these changes the great omentum bulges more toward the right and approaches the duodenal loop as far as the right bor-

der of the mesocolon. Fusion between the right border of the omentum and the loop of the duodenum occurs before this has attained its full size. The extent and time of this fusion seems to be variable and upon this variability may depend the degree of extension of the omentum to the right. There is generally a fusion between the transverse colon and the developing duodenum before the omentum receives an attachment to the colon. This fusion may limit the extent of fusion of the omentum to the right. This early fusion between the colon and duodenum forms the right border of the gastrocolic ligament and accounts for variations in this ligament. It is about this time that the posterior surface of the duodenum becomes fused with the posterior parietal wall. This fusion is relatively late.

The relation of the omentum and the duodenum and the transverse colon in the adult may be explained by an early fusion of the great omental loop to the transverse colon. The growth of the cecal segment of the gut, as well as the lateral thrust it receives from the growth in length of the duodenal loop, serves to carry the whole mass toward the right. In this way the omentum may be carried far to the right with attachments to the hepatic flexure and even to the right parietal wall (Fig. 32). When this occurs there is a fold made up of the right edge of the great omentum extending from the duodenum to the colon and becomes the gastrocolic ligament.

As the duodenal loop extends to the left the free edge of the anterior mesogastrium thickens and is supposed to become the hepatoduodenal ligament though the greater part of this structure is newly formed tissue. The edge of the omentum extends down over the duodenum to the colon and may thus form a continuous membrane forming the hepatocolic ligament. There is no good embryonic excuse for this name. It is only a combination of two folds extending from the liver to the duodenum representing the hepatoduodenal and another representing the right lateral fold of the gastrocolic omentum, the gastrocolic ligament. These folds are unified and intensified by the independent fusion between the colon and duodenum. In this early stage the duodenal loop occupies a higher position relative to the liver than it does in the adult. Receding from the liver, as it does later in life, the traction raises

one or more plicæ, the cystocolic ligament. These may be of surprising size in early embryos. Continued growth must tend to this obliteration for they never retain the size they attain about the seventh month of intrauterine life. It is of interest to note that commonly when these folds are of large size the pyloric region lying below receives the stimulus and becomes excessively

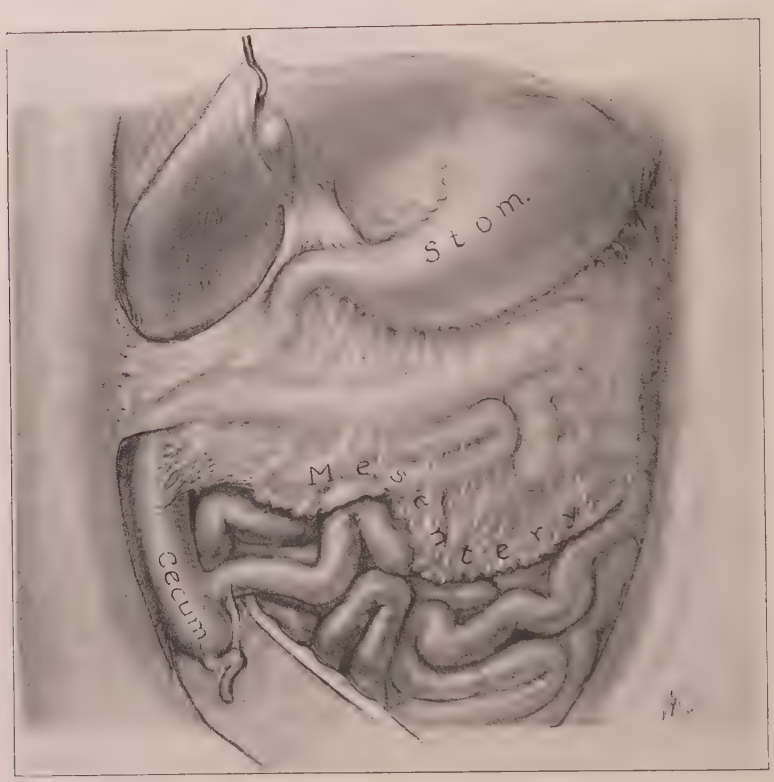

Fig. 32.—The great omentum is fused to the ascending colon and the right parietal wall.

thickened. This state resembles the pyloric stenosis of the newborn and possibly might have become such had they not so early contributed their bit to science.

When the hepatoduodenal region is studied in embryos of from the sixth to the eighth week, the gall bladder is seen to impinge firmly upon the gastroduodenal junction. Plastic material surrounds these structures quite comparable with the exudates formed

in the early stages of wound healing. As the gall bladder is gently raised up, fibrinoid bands regularly extend between it and the duodenal-colic region and along the lateral edge of the great omentum, if that structure has already reached this point. It is easy to understand how some of these attachments may remain permanently and form a sort of meson for the gall bladder. In fact one is surprised that there is so seldom a mesentery between the gall bladder and the duodenum. It would seem plausible that if such a band of fibers can form there, the hepatocolic ligament might be accentuated by the development of new reinforcing fibers between the liver and colon, which were not present in the anlages of the primary mesenteries. One of these bands, the hepatorenal, has received official sanction. There are many others, sometimes observed, not so sanctioned. In some fetuses these bands are found in unexpected places. Such findings have been interpreted as the result of a fetal peritonitis, but I am disposed to doubt the justification for the use of this term in this connection.

An attempt has been made recently to connect the cysto-duodenal ligament with the remains of the anterior mesogastrium. Henle is the only author I have found who implies that the right fold may reach the gall bladder. This must be incorrect, for the direction of the development of the gall bladder is lateral to, and away from, the lower edge of the mesogastrium. Be this as it may, the development of bands in this region is the expression of normal variations and their presence does not obligate the surgeon to hypothecate past pathologic processes, nor warrant surgical procedures for their release. Still less is he warranted in seizing on them as a solace for a wrong diagnosis. The study of this region with its multitude of variations reminds one of hand-made machines, each presents its little variations and are all properly made so long as everything runs smoothly. The surgeon should get away from his conception of machine-made goods with their standardized parts.

Development of the Ileocecal Region.—The concluding remarks of the past paragraph apply with equal force to the ileocecal region as no one has ever seen a "normal" cecum except the one depicted in the textbook of his student days.

Contrary to the schematic cuts with which the literature abounds,

the cecum does not make, in any except rare instances, a grandiose sweep over the middle of the belly to gain its position in the right iliac fossa. On the contrary it impinges against the posterior abdominal wall just below or just lateral to the duodenal jejunal attachment, securing adhesion to the posterior abdominal wall.

The exact point where the cecum becomes attached to the posterior wall both as relates to the gut and the abdominal wall is subject to considerable variation. Usually so far as it relates to the gut the fusion first takes place just above the ileocecal junction but may occur more distal, that is, nearer the sigmoid or may occur nearer the tip of the cecum. In some specimens the first agglutination takes place a centimeter or more from the ileocecal junction. This is perhaps the case in the future mobile cecum. In rare instances no attachment at all takes place and the cecum and ascending colon retain throughout life a complete mesentery.

After the primary attachment has become fixed, the cecum, very broadly speaking, has become retroperitoneal. This expression is misleading unless it is remembered that by peritoneum is understood the layer of slightly differentiated cells out of which the future peritoneum is formed. Then, as the ascending colon elongates, the cecum plows along this relatively undifferentiated tissue to its permanent abode.

In making its journey many variations in detail may take place both on the part of the peritoneum and the omentum. Ordinarily the omentum is influenced only by the position of the hepatic flexure. If, however, the lateral edge of the great omentum should become attached to the cecum while the two are sojourning in the region of the duodenum the more stalwart cecum might drag the omentum to the ileocecal home.

As already noted as the descent of the hepatic flexure of the colon from the liver takes place, plicæ are produced. The most dignified of these is the so-called hepatorenal fold, which however has nothing to do with the kidney but is the index of the former relation of colon and liver. Many other folds are sometimes observed, all bearing more or less historic information in that they bear evidence of early embryonal relations. As the colon descends along the right lateral wall, attachments are secured which when traction is produced by the descending cecum plicæ are produced.

These ordinarily prominent in the fetus, as Flint has emphasized, become more or less obliterated in the adult and become prominent only when disease makes them again prominent. Nevertheless, they remain throughout life, nothing more than evidence of details of development. The head of the cecum may secure attachments with the abdominal wall after descent is completed. In this way, bands extending from the abdominal wall to the cecum and ascending colon are formed. When these form before the descent of the cecum has occurred they are directed from without down-

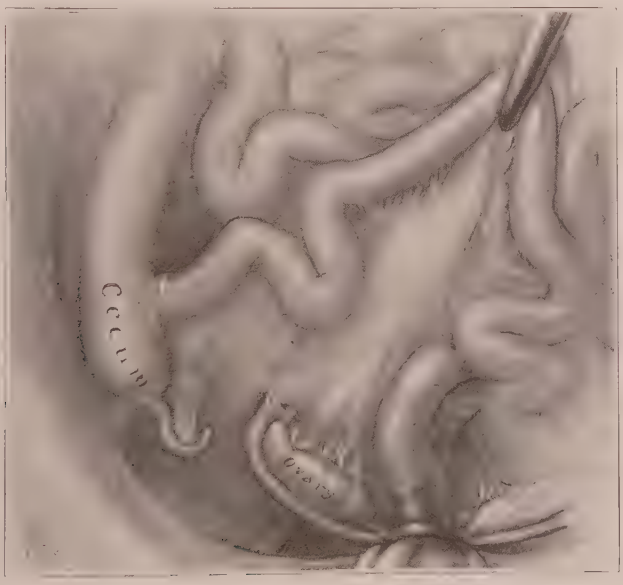

Fig. 33.—The genitomesenteric fold.

ward and inward but when they are formed after the cecum has descended they are directed inward.

As the cecum reaches the site of its permanent abode its posterior surface is devoid of peritoneum. As the ileum enters the gut at this point it must at least at its terminal extremity become retroperitoneal. At the point where it sacrifices its mesentery to reach the cecum a fold exists varying in extent according to the point from which the small gut approaches the large. When it begins high up along the pelvic brim it may be but little marked. When it

approaches from the depth of the true pelvis as it usually does this fold is larger. When traction is made on the free portion of the gut this attachment may be made to appear as plica or band. The mobility of the peritoneum may be so great and Douglas' pouch so shallow that traction on the ileum may cause a fold to appear extending to the broad ligament and even to the ovary (Fig. 33).

In all these regions in the fetus the organs are confined in a rela-

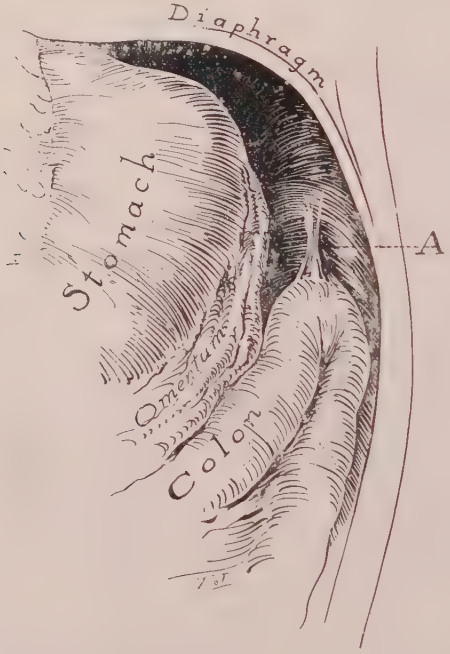

Fig. 34.—The phrenocolic ligament (*A*) already formed while the great omentum is just beginning to develop.

tively compact space. As adult life is approached the liver retracts upward owing to its lessened relative size while the genital and pelvic organs descend. These facts tend to the formation of other plicæ than those already accounted for. In the interpretation of a given fold both factors must be considered.

Development of the Splenic Region.—The disposition of the spleen has already been considered. The flexure of the colon extends high up at this point due to the increase in length after other

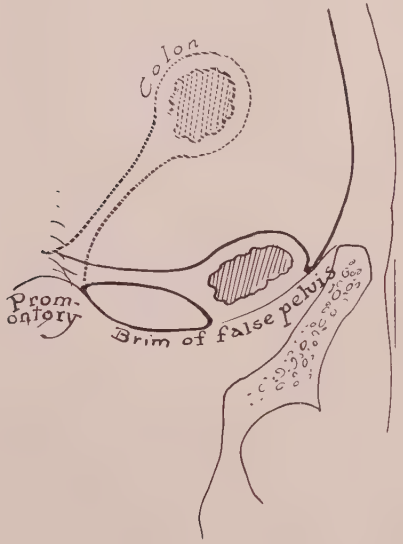

Fig. 35.—Diagrammatic drawing of the fixation of the sigmoid.

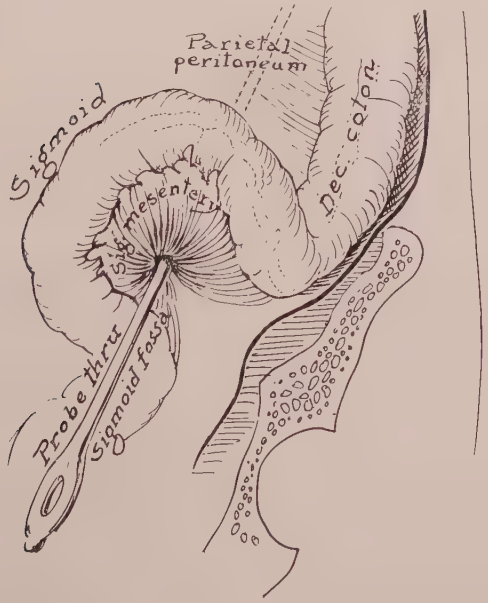

Fig. 36.—Incomplete obliteration of the mesosigmoid leaving the fossa.

parts of the colon have become attached. The splenic angle reaches its permanent place earlier than any other portion of the gut tract and is the most constant in its topographic relations.

Usually the splenic angle of the colon reaches the tip of the spleen while the ascending colon is still possessed of a mesentery. The method of attachment of the gut in the region of the spleen is variable. Sometimes the gut becomes attached to the abdominal wall below the spleen by one or two folds before the omentum reaches this region and sometimes a fold of omentum becomes attached to the abdominal wall before the splenic fold of the gut reaches its final abode. In this event the ascending gut becomes attached simultaneously to the omentum and parietal wall. The final result is a well-marked band sometimes bifid, extending from the extreme angle to the parietal wall (Fig. 34).

Development of the Sigmoid Region. Up to the seventh month the mesosigmoid and the lower portion of the descending colon are free. The entire extent destined to ultimate attachment becomes simultaneously fixed along the posterior surface of the gut (Fig. 35). If a portion of the mesentery fails to become adherent, a sigmoid fossa remains (Fig. 36).

Bibliography

Bochdalek, Jr.: Ueber den Peritonealüberzug der Milz und das Ligamentum pleurocolicum (Beitrag zur Anatomie des Bauchfells), Arch. f. Anat. Physiol. u. wissensch. Med., 1867, 565.

Broesike: Ueber intraabdominale (retroperitoneale) Hernien und Bauchfelltaschen, nebst einer Darstellung der Entwicklung peritonealer Formationen, Berlin, Fischer, 1891.

Froriep: Einige Worte über den Vortrag der Anatomie auf Universitäten, nebst einen neuer Darstellung des Gekröses und der Netze, Weimar, 1812.

Hansen: Peritonaei humani anatomia et physiologia, Berolini typ. Nietachianis, 1834.

Henle: Anatomie des Menschen, ii, Viegel Sohn, Brawnschweig, 1866.

Hennecke: De functionibus omentorum in corpore humano, Goettingae typ. Dieterschianis, 1836.

Huschke: Lehre von den Eingeweiden und Sinnesorganen des menschlichen Korpes, v. Sömmerring's Handbuch, Leipzig, 1844, p. 201.

Lockwood: The Development of the Great Omentum and the Transverse Mesocolon, Jour. Anat. and Physiol., 1883-4, xviii, 257.

Meckel: Bildungsgeschichte des Darmkanals der Säugetiere und namentlich des Menschen, Deutsch. Arch. f. d. Physiol., 1817, iii, 1.

Müller: Ueber den Ursprung der Netze und ihr Verhaltniss zum peritonealsacke beim Menschen aus anatomischen Untersuchungen an Embryonen, Arch. f. Anat. u. Physiol., 1830, v, 395.

SWAEN: Recherches sur le développement du poie, du tube digestif de l'arrière cavité de péritoine et du mésentèri, Jour. de l'anat. et de la physiol., 1897, xxxiii, 32, 222.

TOLDT: Bau und Wachsthumsveränderungen der Gekröse des menschlichen Darmkanales, Denkschr. d. Mathnaturw. Klasse, k. Akad. d. Wissensch., 1879, xli.

Darmgekrose und Netze in gesetzmassiger und in gesetzwurdiger Zustand, Ibid., 1889, lvi, 1.

Uber die Geschichte der Mesenterien, Verhandl. d. anat. Gesellsch. in Goettingung, 1893, 1.

TREITZ: Hernia retroperitonealis. Ein Beitrag zur Geschichte innerer Hernien, Prag. F. A. Credner, 1857.

ZOERNER: Bau und Entwicklung des Peritoneum, nebst Beschreibung des Bauchfelles einiger Edentaten, Halle a. S. Gebauner-Schwetschke, 1881.

CHAPTER IV

GROSS ANATOMY OF THE PERITONEUM

General Consideration.—The peritoneum is the membrane which lines the abdominal cavity, and covers the organs contained in it. It also envelops the ligaments which project into the lumen of the abdomen and lines the depressions in which the organs lie. A comprehension of its arrangement implies a knowledge of organogenesis and topographic anatomy. A consideration of its topographic relations must take into account both the general average arrangement in the adult state and also those variations which are within the normal range, yet may simulate abnormal conditions. The factors of importance to the operating surgeon will receive particular attention, as will those conditions which simulate the products of past disease.

A general survey of the entire peritoneum is desirable as a preliminary to the closer examination of the special regions. Its general relations are best emphasized by saying that, if the operator were skilful enough, it would be possible to dissect off the peritoneum from all the underlying structures and from the structures which it envelops, and thus secure a complete sac. In its sac-like character it resembles the skin, for one can imagine the entire cutaneous surface removed, also forming a complete sac, save for a few orifices. The ancients demonstrated this nature of the skin by removing the hide from animals and using them as receptacles for holding and carrying water. Theoretically, the peritoneum might be employed for a similar purpose. This emphasizes the fact that the individual is interposed between a cutaneous sac and the serous sac. Both of these sacs are folded about irregularities of surface, outside and inside. A clear understanding of the relation of these two surfaces goes a long way toward clarifying the true function of the peritoneum.

Though the peritoneum presents a continuous surface, it is advantageous to divide its area into the parietal, that portion lining

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the wall of the abdomen, and the visceral, that portion covering the intraabdominal organs and their mesenteries. While this division is arbitrary and artificial, it facilitates the presentation of many very complex, though practical, topographic relations. Similar practical considerations make it desirable that the variations in its attachment to the underlying tissue, notably the ligaments and adipose tissue, be accorded separate consideration. It is the variations in this subperitoneal tissue, both in amount and character, which account for the varying degree of mobility of the various organs, as well as the disposition of foreign accumulations to extend with greater facility in certain directions. It is impossible here also to repress reference to the analogy between the peritoneum and cutaneous surface.

Extent of the Surface.—The extent of surface the peritoneum has to cover, together with the undulations and reduplications it undergoes, makes it by all odds the most extensive serous surface of the body. It may be said that the opinion generally held, that its area is coextensive with that of the surface of the body, is in the main correct.

In order to determine the accuracy of this general notion I measured twenty bodies in the deadhouse. These persons had died of accident or of acute disease in which emaciation had not taken place. The average of these measurements gave a total peritoneal surface of 3,268 square inches (21,986 sq. cm.) and a total cutaneous surface of 3,436 square inches (22,167 sq. cm.). The average height of these bodies was 5 feet and 8 inches, the average weight was estimated at 160 pounds. Wegner, as a result of very careful measurements, reaches a total somewhat less than that of my own. He gives the surface of the peritoneum as 17,820 sq. cm. (2,663 sq. in.) and that of the body as 17,502 sq. cm. (2,712 sq. in.). The height and weight of the cadavers from which these data were obtained are not given.

The tensile strength and elasticity of the peritoneum vary in different regions. Hyrtl quotes Scarpa as saying that a stretched piece of peritoneum will bear a weight of 15 pounds, and Huschke who found it could bear 50 pounds. Obviously the strength depends upon the amount of subperitoneal connective tissue included. My own experiments gave even less than Scarpa's for the part

free from subperitoneal tissue. The meshes of the mesentery, for instance, often bear less than one pound.

Relation to Its Environment.—Before proceeding with the discussion of the peritoneum it is worth while to note that, coextensive with the parietal peritoneum, there is a fascia which lines the structures composing the abdominal wall. This is composed of the rectus fascia, the transversalis fascia, the quadratus fascia, the fascia of the pelvis, etc., and was designated by Luschka the fascia endoabdominalis. Collectively they form a strong fibrous encasement for the whole abdominal cavity. It forms a strong support for the intraabdominal organs and yet permits of free movement of the muscles covering it. It is to this fascia that the peritoneum bears an intimate relation when not separated by the interposition of viscera or adipose tissue. This fascia may be compared with the fascia covering the skeletal muscles in that it is essentially sustentacular. The endoabdominal fascia is doubly worthy of consideration for the peritoneum is so closely attached to it in certain regions that surgeons, when discussing surgical technic, are wont to regard it and the peritoneum as one structure. There will be frequent occasion to allude to this relationship when dealing with surgical topography.

The parietal peritoneum is held in apposition to the endoabdominal fascia by connective tissue. In many regions the attachments are loose, admitting of ready separation; in other regions they are so close that separation is accomplished only by sharp dissection. It is loosely attached in part or wholly free from retroperitoneal organs when these organs are required to expand and contract in the performance of their function, or if these organs require mobility in order to accommodate themselves to the movements of other organs which change their volume. It is particularly loosely attached in the posterior regions, where the kidney must move in response to the action of the diaphragm or to the necessary movements of the colon, and in the lower anterior region about the bladder in order to accommodate the altering capacity of that organ. Where no such movements are required the attachments are closer, depending on its elasticity to admit the necessary change in volume of the organ, as over the gut tract and the intraperitoneal solid organs. Over the latter the peritoneum does

not anticipate the possibility of any considerable change in volume, as is noted by the sparseness of the elastic fibers. Where no change in position or volume of the underlying organ is required, the peritoneum is in immediate apposition to the underlying structure. This is marked, for instance, at existent or potential openings, over the recti muscles, about the umbilicus, about the inguinal region, and about the central tendon of the diaphragm.

The parietal peritoneum undergoes certain elevations and depressions which are of importance, aside from those necessary for the accommodation of viscera. The depressions may be associated with vestigial openings, which once permitted the passage of organs to the outside of the cavity, and remain as potential openings for further mischief, or partially to separate portions of organs to permit them better to accommodate themselves to their assigned spaces. The elevations exist to accommodate vestigial structures or necessary supports to retain the organs within the range of mobility demanded by their function. These irregularities are of anatomic, often of phylogenetic, interest. Their surgical interest lies in their importance as topographic landmarks and as frequent sites of surgical lesions. Among the more important elevations may be mentioned the round ligament of the liver, the hepatoduodenal, the splenic, and the broad ligaments of the uterus in the female, etc. In the case of some of these ligaments the peritoneum exists virtually as mesenteries to these structures. It is often a matter of arbitrary election, therefore, in what class many of these elevations are placed, whether ligaments or plicæ, etc. In such designations it is important to note that the name "ligament" applies to the structures beneath the peritoneum, and not to the peritoneum itself. The peritoneum never exercises a sustentacular function.

Certain viscera, such as the colon, bladder, etc., are interposed between the peritoneum and the endoabdominal fascia, being but partly covered by the former. The colon is intraperitoneal in some regions, partly so in some regions and not at all in others, so that it is in part an extraperitoneal and in part an intraperitoneal organ. The spleen also exhibits individual variations in this regard.

The foregoing remarks are sufficient to indicate that there is

great variation in the relation of the parietal peritoneum to the endoabdominal fascia. The knowledge of this relationship is of so great importance that it is desirable to accord to it an intensive anatomic examination.

General Relations of the Peritoneum.—Before entering into such

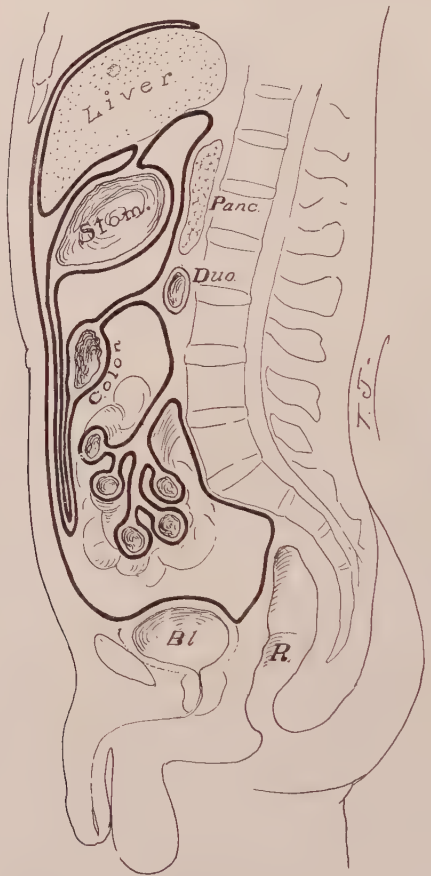

Fig. 37.—Longitudinal section of the body showing the general relation of the peritoneum (heavy black line) to the underlying structures.

study it will facilitate the understanding of the special regions to present a general outline of the relation of the peritoneum to the abdominal wall and its contained structures. In making a cursory examination we may follow the description of the anatomists

and begin arbitrarily at the umbilicus, the point of departure usually selected, and trace it in turn upward, downward, and later-

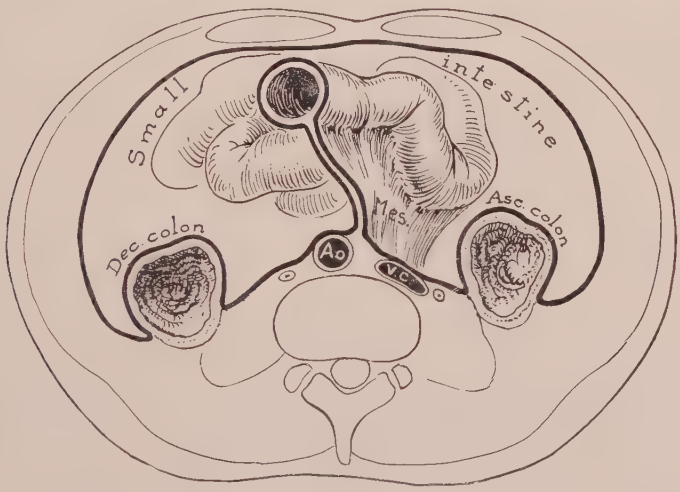

Fig. 38.—Cross section of the body at the level of the umbilicus showing the general relation of the peritoneum (heavy line) to the underlying structures.

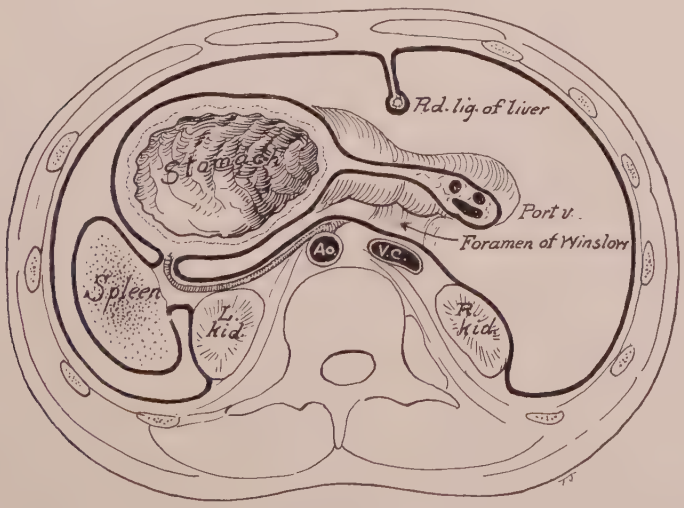

Fig. 39.—Same as Fig. 38 at the level of the foramen of Winslow, showing peritoneum (heavy line).

ally. We may employ the diagrammatic drawings presented by the textbooks to facilitate the presentation.

Beginning at the umbilicus (Fig. 37), and passing downward along the anterior abdominal wall, the peritoneum is found to lie

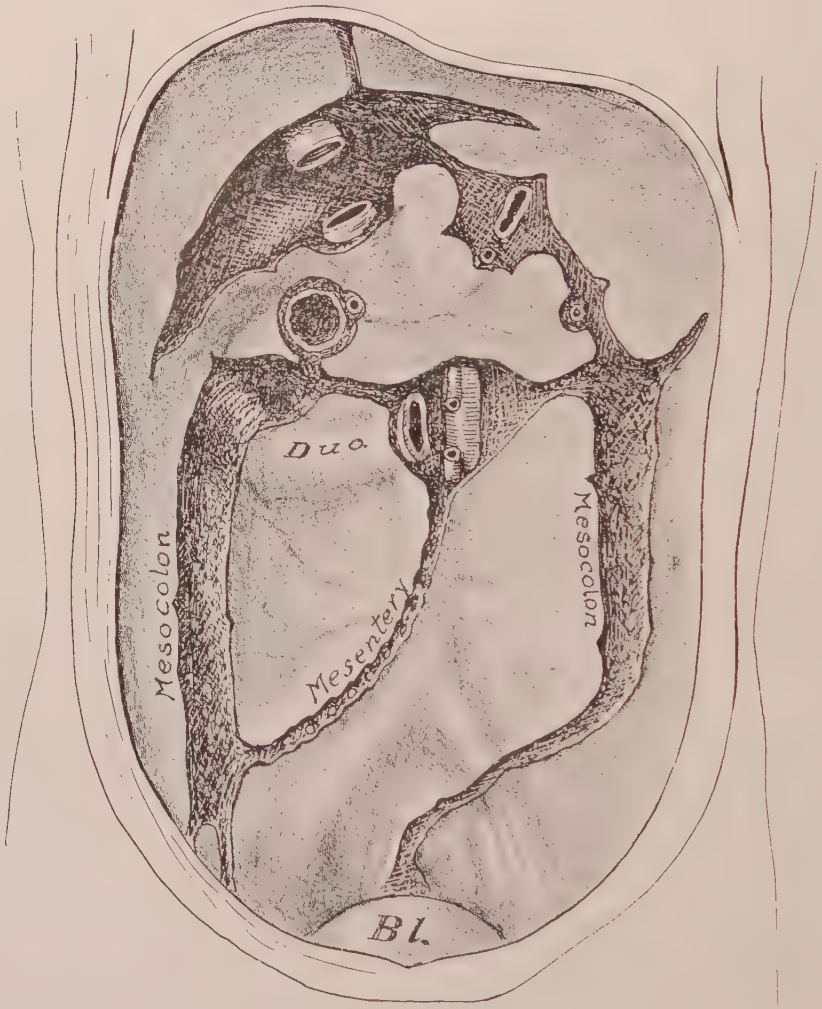

Fig. 40.—Diagrammatic drawing showing the extent of interruption of the parietal peritoneum by the interposition of viscera. (Redrawn from Meckel.)

at first closely attached to the fascia of the transversalis and recti muscles. Near the pubis it is separated from the fascia by a pad of fat, which is interposed between it and the recti muscles. De-

scending over this, it secured a loose attachment to the pubis and is reflected over the bladder. It passes over this organ to the depth of Douglas' pouch in the male; but in the female it rises again to cover the uterus before descending into Douglas' pouch. From this point it ascends along the posterior wall of the pelvis and securing a close attachment to the sacrum, it passes upward, being separated from the spinal column only by the great vessels and the areolar tissue surrounding them. Its ascent is interrupted near the midline of the abdomen by the root of the mesentery. Escaping this interruption, it passes upward over the transverse portion of the duodenum, to be again interrupted by the mesocolon. From the transverse mesocolon it ascends under the stomach and reaches the under surface of the liver as far as the falciform ligaments. If we ignore for the moment the audacious intrusion of the liver we may begin at the under surface of the diaphragm, and follow it down to the anterior abdominal wall, and hence to the point of beginning as the surveyors say, namely, to the umbilicus. In this last lap it is in close association with the fascia of the recti muscles, being interrupted only by the round ligament of the liver.

As noted, the ascent was interrupted by the mesentery supporting the small intestines, the mesentery of the transverse colon, and the protrusion of the liver with the lesser omentum and stomach suspended below it. The stomach is further united by a membrane with the transverse colon. As a result of this an accessory cavity is formed, the lesser peritoneal cavity. As seen on longitudinal section, therefore, the abdominal cavity is divided by the projection downward and forward of the mesentery of the small intestine, the mesocolon, and the lesser omentum.

In tracing the relation on transverse section by passing laterally to the right from the anterior midline, the peritoneum is seen to follow the abdominal wall in close relation with the transversalis fascia to near the anterior border of the quadratus lumborum muscle (Fig. 38). At this point it is raised from the fascia by a layer of fat. Curving medially it passes over the summit of the colon and is then deflected forward over the body of the psoas muscle. It then passes toward the median line until interrupted by the root of the mesentery, and takes up its course beyond the median line. Extending from this point it proceeds over the de-

scending colon to the edge of the quadratus muscle, from whence it follows closely attached to the left parietal wall, which it follows to the point of beginning.

At higher and lower levels this relation is somewhat modified by the interposition of various organs. In the region of the pancreas, instead of being interrupted by the root of the mesentery, it passes the body of this organ. Still higher it passes through the foramen of Winslow into the lesser peritoneal cavity as far as the spleen (Fig. 39). It returns along the posterior surface of the stomach to the hepatogastric ligament, over which it passes to reach the anterior surface of the stomach. From here it loops over the spleen, forming its capsule, and ascending along the lateral and anterior abdominal wall, it reaches the midline.

When one views the recently opened abdomen with the viscera in place, one gains the impression that a considerable portion of the posterior parietal peritoneum is interrupted by viscera. When the viscera are carefully removed, however, in hardened subjects, it is readily seen that the area thus occupied is relatively small (Fig. 40). Even this extent of surface may be lessened by the formation of a true meson for the ascending colon. In that event the attachment of the colon, like that of the small intestine, is represented by a narrow line. An appreciation of the relatively small area of the attachment of the organ to the posterior abdominal wall aids much in understanding the anatomic basis of the capacity of the abdominal viscera to accommodate themselves to the movements of the body or to the shifting of position of the viscera to accommodate each other.

The Retroperitoneal Tissue.—Before taking up a more intensive study of the relation of the parietal peritoneum to the parietal wall and to the viscera, it will be profitable, on the meager basis already presented, to consider the retroperitoneal tissue and the relation of the peritoneum to it.

The retroperitoneal connective tissue varies much in amount and structure, but is, for the most part, loose areolar tissue encapsulating fat in the degree native to the general nutritional state of the individual. Where the attachment of the peritoneum to the fascia is close there is a direct fibrous union with little or no interposition of fat. In some situations in fact the peritoneum

is so closely attached to the underlying structures that interposition of fat never occurs. This may be noted particularly about the umbilicus and the central tendon of the diaphragm. It is likewise sparse on the anterior wall lateral to the umbilicus. In these regions the association between the peritoneum and the underlying fascia is exceedingly delicate, too delicate, in fact, to admit of independent manipulation in practical surgery.

On the other hand, where mobility of extraperitoneal organs is demanded the connective tissue is abundant. Above the pubis the peritoneum is separated from the recti by a generous pad of fat. The same is true of the iliac fossa, and particularly so on the posterior wall in the region of the kidneys and about the great abdominal ganglia. This abundance of the subperitoneal tissue permits a free mobility of the peritoneum over the underlying structures; and, when the underlying structures are movable, it permits their movement independent of the peritoneum. It provides structures not movable with a protection against injury.

In each instance the character of tissue is such as best to adapt it to the purpose. It is loose where mobility is required, but in those regions where it acts as a protecting pad it is exceedingly resistant to efforts of the dissector to remove it. This quality is most marked about the sympathetic ganglia and the adrenals. It is interesting to note that the latter type of fat fluctuates in amount but little with the varying state of nutrition of the individual.

While abundance of subperitoneal tissue secures physiologic mobility and protection to the organs which it surrounds, this is not an unmixed good for the reason that pathologic emanations from the extraperitoneal organs receive full play in this loose tissue, just as infection spreads unhindered in subcutaneous areolar tissues. The surgeon is favored in that the looseness of the attachment of the peritoneum permits the ready performance of operations upon organs lying in these regions. Operations, for instance, on the duodenum, the kidney, and the ureters are particularly facilitated by such loose attachments.

The production of hernia is not facilitated by the looseness of attachment of the peritoneum, as is sometimes stated. In most situations where the peritoneum is loosely attached the protecting wall of muscle and fascia is particularly strong. At the common

site of hernias, as in the inguinal canal and the umbilical region, the peritoneum is closely attached to the underlying fascia. In recurrences after inguinal and umbilical herniotomies the peritoneum lining the sac is very much attenuated. It seems probable that in the latter variety of hernia the peritoneum is ruptured, and a new-formed membrane is produced to line the sac. This opinion is supported by the fact that some of the accessory pockets in large umbilical hernias are devoid of basement membrane. To say, however, as Petit does, that all the peritoneum of umbilical hernial sacs is newly formed, is, no doubt, overstating the facts, for in the smaller hernias the basement membrane is continuous with that of the abdominal cavity. It is only in postoperative hernias that the normal looseness of the peritoneal attachments may be said to facilitate the production of hernias.

For the purpose of study, a variety of special regions in the retroperitoneal space may be distinguished, though there is no sharp dividing line. The upper abdominal, the renal and the iliopelvic, and the prevesical are of the most conspicuous importance.

The extent of these spaces may be most impressively visualized by the injection of a thin solution of plaster of Paris, as suggested by König for the mediastinum. After these masses harden, their outlines are definitely delineated. The avenues of least resistance to cumulative pressure are best demonstrated by the injection of methylene blue under moderate pressure. The surgeon of experience will have had occasion enough with pus accumulations to appreciate the more important lines of least resistance, while the junior surgeon may anticipate experience by experimental work here suggested. The remarks to follow are based on information gained by all three methods above noted.

The Retroperitoneal Spaces of the Pelvis.—The connective tissue of the true pelvis is distributed chiefly about the front and the sides of the bladder, in the broad ligaments, and about the pelvic sigmoid. Because of the frequency with which abscesses occur in this region as complete a knowledge as possible of these spaces is desirable because it is only by considering these factors that the direction of extension can be anticipated.

The most important regions are those above the bladder, lateral to the bladder, beneath the bladder about the seminal vesicles,

about the pelvic colon, and particularly those spaces situated in the base of the broad ligament.

The Prevesical Space.—The degree of separation of the peritoneal flexure from the pubis varies widely in different individuals and varies from time to time in the same individual, dependent upon the fatness of the individual and the state of distention of the bladder. This space continues below the symphysis, being covered above and behind by the peritoneum, and bounded anteriorly by the vesical fascia. (The fascia vesicalis of Charpy, La gaine des muscles deposits et la cavaté prévésicale. In: *Études d'anatomie appleqúe*, Paris, Bailliere, 1892, p. 183.) The loose connection of the peritoneum extends as far as the puboprostatic ligaments anteriorly; and laterally, as far as the ureters. This space was first described by Retzius by whose name it is commonly known.

Usually about the semilunar line of Douglas, the peritoneum loosens its hold on the rectus fascia, and comes to include a layer of fat (Fig. 41). The degree of separation is usually dependent on the general fatness of the individual. In corpulent persons it is not unusual to find large masses of fat between the fascia and the peritoneum, sometimes extending upward to near the umbilicus. This fat is often separated into planes, so that young operators sever these one at a time, believing each to represent the peritoneum. The final layer may be very thin, being formed of only the basement membrane and the endothelial cells, which may tear on traction. It is easy under these conditions inadvertently to perforate the peritoneum above the bladder when doing a suprapubic prostatectomy. This is likely to be true when the hypogastric remnants or urachus are prominent.

As the pubis is approached the space between the peritoneum and the fascia widens. Near the level of the pubis, it is deflected over the summit of the bladder, and becomes intimately associated with the wall of this viscus 4 to 5 cm. from the pubic bone when the bladder is empty; when the bladder is filled the space is greater. For this reason it is customary for surgeons to fill the bladder with an indifferent fluid before opening it. In children the bladder stands proportionately higher than in adults. It is higher also in obese persons, and in persons whose bladders are

often distended to an extreme degree. The degree of separation, for instance, in old prostatics with chronically distended bladder may reach 10 cm. or more.

The practical importance of this point makes it worthy of a more detailed consideration. Studies made on the cadaver are unsatisfactory because of the postmortem rigidity of the tissues. In

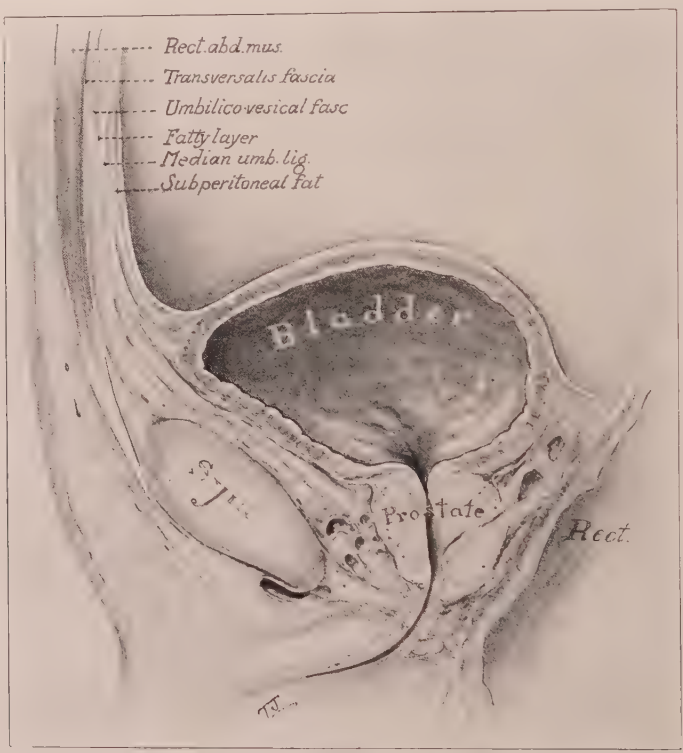

Fig. 41.—The relation of the peritoneum to the space of Retzius, the bladder, and the structures composing the anterior abdominal wall. (Modified from Waldeyer.)

order to overcome this difficulty I have kept this point in mind when operating on patients whose bladders were spontaneously distended with urine. These observations indicate that the pubovesical fold recedes with equal distention more in the living than in the cadaver. In the living when the bladder reaches nearly to the umbilicus the fold may be elevated to as much as 8 or 10 cm.

from the pubic bone. Lesser degrees of distention raise it in reduced proportion. Chronically distended bladders, as in incontinence of retention, raise it more than acutely distended bladders, as those for instance following traumatic ruptures of the urethra. In general it may be stated that the peritoneal fold rises half the distance to the fundus of the distended bladder. This rule applies, of course, only to the moderate distention observed on the operating table. In extreme cases where the bladder extends over the promontory of the sacrum, like a pregnant uterus, this obviously does not obtain.

In some instances the separation, instead of beginning at the semilunar line, begins immediately below the umbilicus. In 1 or 2 per cent of cases, on the other hand, according to Waldeyer the peritoneum follows the rectus fascia closely to the top of the pubic bone, to which it receives attachment. The proportion above noted seems to me to be excessive. I have looked for this relation for many years, but have never found it either in the dead or the living. The importance of this relation, as may be inferred, lies in the fact that, when it exists, a suprapubic puncture or incision into the bladder would traverse the peritoneal cavity.

The loose relation of the peritoneum, as above described, owes its practical importance to the fact that in operations on the bladder it is usually desirable to avoid opening into the peritoneal cavity. The above description defines the area which the surgeon may ordinarily depend upon as a working field. The union of the peritoneum to the underlying structures is seldom so close but that a separation in various directions can be accomplished without injury to it. It is particularly easy to extend the available space upward nearly to the umbilicus, especially when the urachus is persistent. (Compare Disse, Beiträge zur Kenntnis der Spalträume des Menschen, Arch. f. Anat. u. Entwiklugsgesch., 1889, Suppl. Bd., 22.)

The practical importance of the relations of the peritoneum in this region in reaching the apex of the bladder for the purposes of drainage or for purposes of the removal of the prostate, is well understood, but often sufficient consideration is not given it in urinary infiltrations, nor as a possible site of hemorrhagic ex-

travasations in extensive operations upon the ureters or after severe injuries of the pelvis. It is readily available for extraperitoneal drainage in all of these conditions.

The Perivesical Space.—The fat-filled space in front of the bladder above discussed is continuous laterally on either side and downward to the base of the bladder (Fig. 42). This space is

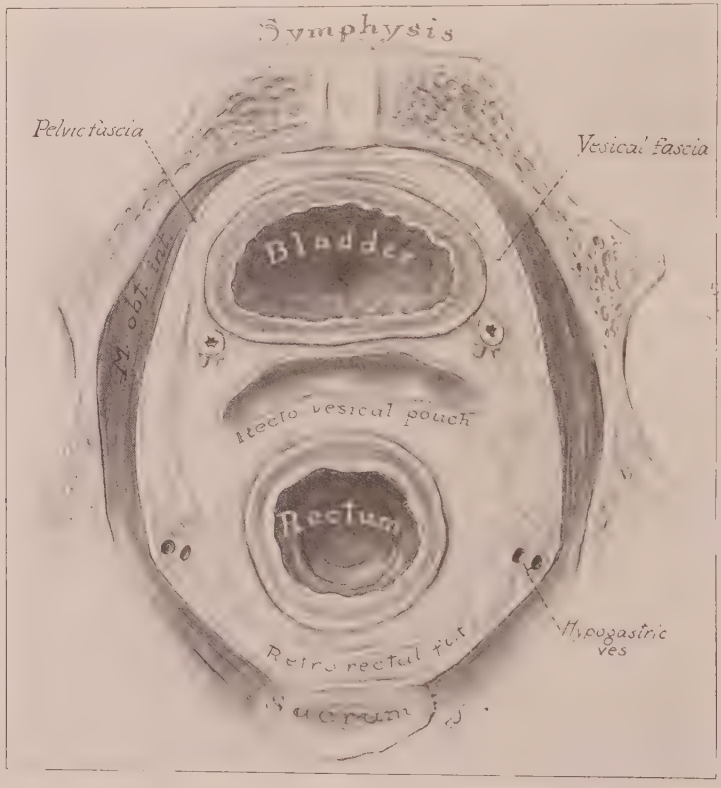

Fig. 42.—Cross section of the pelvis near the bottom of Douglas' pouch.

filled with areolar and fatty tissue, depending in amount on the general adiposity of the individual. This tissue adapts itself to the varying states of distention of the bladder. It extends to the lateral wall of the pelvis over the hypogastric vessels, and the round ligament in the female, to reach the pelvic brim. This arrangement makes easy access to the base of the bladder for the surgeon

in his operative endeavors, but affords equally ready access for urinary extravasations and purulent exudates. The lateral confines of this space is the pelvic fascia, and it terminates at its reflection at the junction of this fascia with the terminal gut.

The importance of this space as an avenue for the approach to the terminal ureter is now generally recognized by surgeons, and it is now being exploited as an available route for attack on the seminal vesicles.

Surgeons, as a rule, do not appreciate that it is easy to reach abscesses in the depth of pelvis by this route. By separating the peritoneum from the side of the bladder and the anterior pelvic wall until the depth of the pelvis is reached, an abscess can be caused to drain extraperitoneally, which otherwise would require a transperitoneal drainage. This is particularly important in the male, and in young females in whom vaginal drainage is difficult and objectionable. This is true whether the abscess arises from subperitoneal infection in the broad ligament or seminal vesicles or from intrapelvic infections, as from the appendix, which have become walled off. That broad-ligament abscesses may elevate the peritoneum, and point in the region of the pubic eminence, has been sufficiently noted; but that this favorable circumstance for drainage may be anticipated seems to have been overlooked.

The Periuterine Spaces.—Within the broad ligament there is a limited amount of loose areolar tissue, which extends from the uterus to the body of the ischium. This space is limited to the base of the broad ligament, and is separated from the space on the middle portion of the broad ligament by fascial planes, which loop over the uterine artery and loose themselves in the pelvic fascia below. It terminates at the body of the ischium. It is difficult to demonstrate these planes by sharp dissection, but they may be demonstrated by the injection methods above mentioned and by microscopic section. Clinical experience confirms the existence of such a space. Infection in this space from indurated masses which have their chief bulk in the region of the ischial body and because of their firm attachment, may simulate neoplastic outgrowths from this bone. When such infections liquefy they tend to point toward the ischial eminence, but they can be reached effectually below.

That portion of the cellular tissue of the broad ligament above the uterine artery is looser in texture than that above mentioned, and is directly continuous with the space above the pelvic brim. Accumulations in this region tend to travel in this direction, and appear just above Poupart's ligament or upward and outward as far as may be required to accommodate their bulk. They may reach the diaphragm by passing in front of Gerota's capsule. By virtue of these connections accumulations may fill the entire space from the broad ligaments over the entire iliac fossa as far down as Poupart's ligament and upward as far as the paraduodenal region. Infections do not tend to spread from the iliac fossa to the broad ligament, however. The fact that it is possible to loosen the peritoneum in front of the kidney by injecting from the iliac fossa, while the iliac fossa can not be injected from the perirenal space, is due to the fact that there is a secondary layer of subperitoneal tissue which passes below the kidney and becomes attached as already explained. Injections from below separate these layers, while injections made above are made through all the layers because of their close apposition. By virtue of the loose attachment of the peritoneum in this region, exudations in the broad ligament extend over the border of the true pelvis into the iliac fossa. In this manner broad ligament infections point above Poupart's ligament, and may extend behind the kidney. Hemorrhages into the broad ligament may extend into this fossa even as far as the diaphragm, as occurred in one of my cases. A similar case is recorded by Boldt.

The Perirectal Space.—There is a sparse amount of retroperitoneal tissue about the pelvic sigmoid (Fig. 42). It is continuous with the mesentery above, and the pararectal fossa below the levator muscle. For a considerable segment of the gut the peritoneum is attached closely to its muscular coat, but toward the sacrum it diverges, including within its folds a variable amount of areolar tissue. Accumulations in this space tend to descend along the wall of the gut, and appear in the pararectal fossæ below the levator muscle. In some instances they tend to follow the iliac crest, and point in the region of Poupart's ligament.

The Retroperitoneal Space of the Renal Region.—The space behind the peritoneum in the region of the kidney may well be di-

vided into three regions (Fig. 43); that in front of the kidney capsule, that posterior to the kidney capsule, and that within the capsule itself. The anterior space corresponds to the lateral and downward extension of the space next to be considered. Laterally this space extends as far as the lateral border of the quadratus

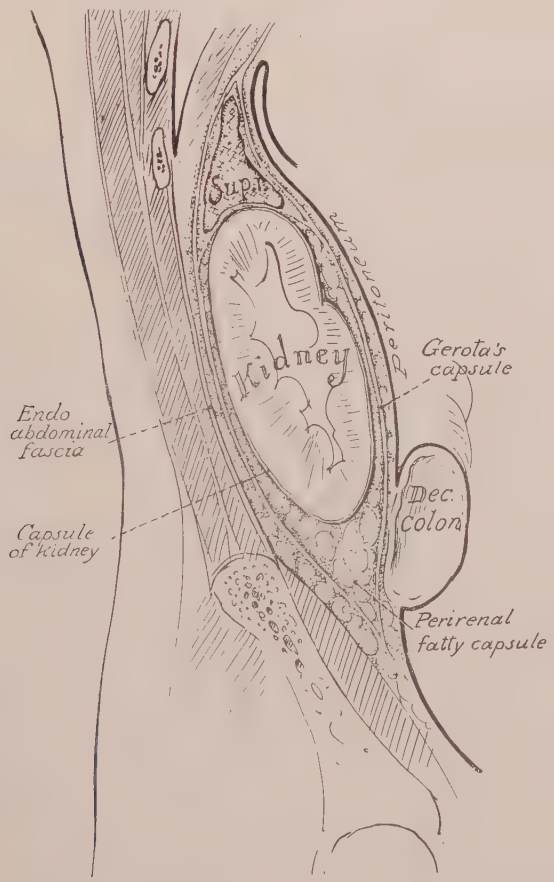

Fig. 43.—The relation of the peritoneum to the perirenal spaces. Longitudinal section.

lumborum. It is represented by a thin layer of loose tissue in front of the capsule and behind the peritoneum. Gerota states that the anterior layer of the capsule is attached to the peritoneum. It does seem so in the cadaver, but the very loose character of

this attachment is easily demonstrated in the living subject. This line of cleavage likewise may be demonstrated in the cadaver by the use of methylene-blue injections. This space has no lower limit. It is this anterior space that crosses the midline. The midline does not form an absolute limit, but the peritoneal attachment is closer in the region of the great vessels; and accumulations under slight pressure tend to become limited at this line. Greater pressure, for instance, that produced by the rupture of an aneurysm, may strip the peritoneum from the vessels for some distance.

Behind this lies the perirenal tissue encompassed by Gerota's

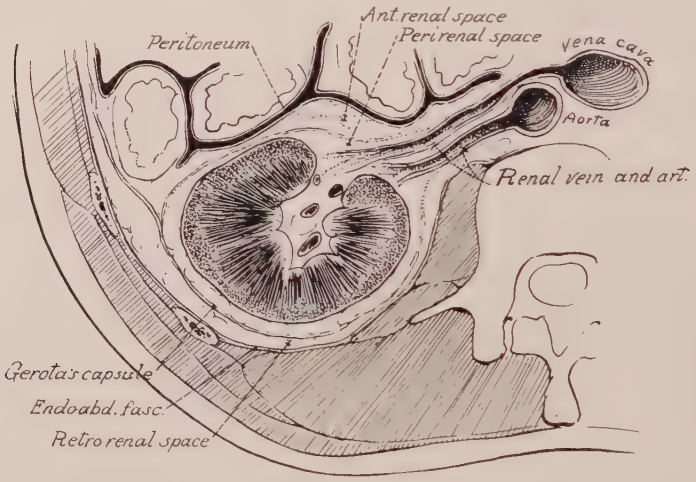

Fig. 44.—Same as Fig. 43. Transverse section.

capsule. This capsule by fibrous extension reaches the fibers from the opposite side along the midline. This tissue is particularly abundant along the renal vessels (Fig. 44), and below a limb extends along the ureter. This fascia is less well marked below the lower pole of the kidney, but can usually be well demonstrated, particularly in stained sections. The continuation of this fascia into that in front of the kidney at both the upper and lower poles explains why perirenal infections are so little prone to extend upward and downward, but instead form globular masses producing prominences below the liver and in the flank. The heavier bands of fascia, demonstrable by gross dissection extend downward as

described by Gerota. Longyear has traced a continuation to the cecum.

Between Gerota's capsule and the quadratus fascia is a space more or less continuous with the anterior space. Above it extends behind the suprarenal capsule as far as the diaphragm. By following this route the infection may reach the space between the liver and diaphragm by rupturing through the attenuated peritoneum at this point. This is the direction traveled by pus in the truly retroperitoneal appendices, and those which have been made such by peritoneal adhesions.

Below this space is continuous with the iliac space (Fig. 45). This space is one of great importance in clinical surgery also because the source of the infection may be determined in a measure by considering the direction of its extension. Infections from the pelvis are most apt to extend anterior to Gerota's capsule between it and the peritoneum, and may be reach the perivertebral space. Infections, as above noted, arising from the kidney proper, at first at least, play their course within Gerota's capsule, and distend it in a globular fashion. After the infection perforates this membrane the retrorenal space becomes involved. The retrorenal space usually receives its primary infections from the appendix. In infections in this region, if the genesis is properly interpreted, dissemination may be anticipated by the placing of a drain up in this space.

The separation of the various spaces above discussed is not absolute. In severe or neglected cases any or all of this territory may become involved, and from a single abscess. The importance of individualizing lies early in the infection before such dissemination has taken place. In less virulent infections there is a certain limitation because of the paucity of pressure and the resistance offered by the reactive processes. In such instances the infection may remain confined to one of these regions for a long time.

The Retrorenal Space of the Upper Abdomen.—In the upper abdominal region an indefinite space may be defined extending upward beyond the transverse mesocolon to the diaphragm. It extends above the kidneys taking in the suprarenal spaces (*A* Fig. 45). It extends downward as far as the first portion of the duodenum, its lower limits being formed by this structure and the

root of the mesentery. Laterally the lower limits are anatomically less definite. This limit seems in a measure to be defined by Gerota's capsule; at least, there are fascial planes which include the nerve trunks going from the large ganglia to the suprarenal capsules. Below the vessels to these organs lie in this fascia. These planes travel inward, and are lost in the capsules of the large ganglia on the sheaths of the great vessels. The extent of this space varies greatly, but the area of chief importance lies between the duodenum and the suprarenal capsule, and includes the tissue surrounding the vena cava (*B*, Fig. 45). The planes of this space are made out with difficulty in gross dissection, but they are easily demonstrated by microscopic sections of small fetuses. By the injection of masses of plaster of Paris in this region these areas are usually made out with distinctness.

These fascial planes are best understood by considering the embryologic development. They have their anlage in the primal serosa of the abdominal cavity. When the duodenum becomes attached fascial planes are developed, thus forming the roof of the lower boundaries of this space. Laterally the extent of this space is dependent on the position of the kidney and suprarenal gland. When these organs lie close to the bodies of the vertebræ the space is proportionately larger. The space is limited above by the close attachment of the peritoneum to the crura and the muscular parts of the diaphragm. The peritoneum of Morris' pouch lies above it.

The clinical importance of this space lies in the fact that infections in the space frequently occur by extension from impending perforations of ulcers in this region and from suppurations of the head of the pancreas. In perirenal infections also, abscesses may occur, which, unless specifically sought for in this region, may fail to be drained. Long-continued infection in this region may cause a cicatricial adhesion between the capsule of the kidney, the suprarenal capsule, and the duodenum, so that, in a secondary nephrectomy, a portion of the suprarenal capsule may be inadvertently torn away, or the duodenum opened into. The close proximity of these structures is apparent in *A*, Fig. 45. It was these accidents, and failure to discover abscesses in this region before autopsy in two cases, that stimulated me to a special study

of this region. In one of these cases there was an ulcer of the duodenum perforating retroperitoneally and simulating at first a pyelitis, and subsequently a perirenal infection, which a flank drain did not wholly relieve. The other case was caused by a suppurative pancreatitis. Primary drainage produced only tem-

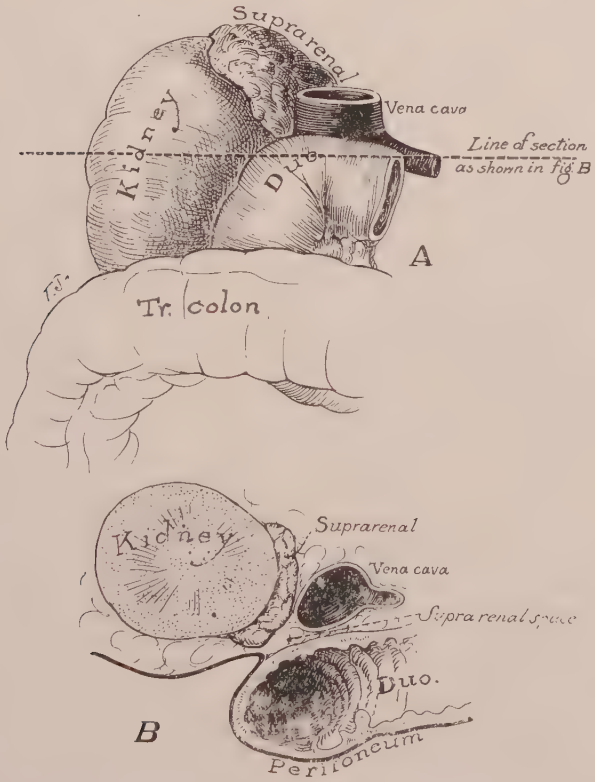

Fig. 45.—*A*. Relation of the viscera of the nephroduodenal region, peritoneum removed. *B*. Cross section of the same region showing the space between the kidney and duodenum.

porary improvement. A flank abscess in the space above described should have been diagnosticated.

Parietal Peritoneum.—Having reviewed, in a general way, the relation of the parietal peritoneum to the tissue upon which it rests, a minute study of the topography of the various regions is in order. It is necessary to study both the structures upon which

it lies and the organs and structures enveloped by it. With a knowledge gained by a study of the development of these regions, we can understand how these various relations come about. The work of conducting such a study is much facilitated by examining a region at a time, depending on our own superficial knowledge of the whole already possessed to keep these separate intensive studies in harmony, so that, when the entire study is complete, our preliminary superficial survey will have become a composite picture of the details of the whole.

In conducting such an intensive study, it is profitable to examine together the various parts which form a region of practical importance, whether such parts fall into definite anatomic regions or not. Thus the pelvic peritoneum, the peritoneum of the renal fossa, the peritoneum of the duodenopancreatic region, and finally, the hepatophrenic and allied regions, may be considered in turn. Such a division is actuated by the desire to consider these regions as entities, which, either because of the relation to extraperitoneal organs prone to disease or the relation to intraperitoneal organs frequently the seat of disease, makes a knowledge of that particular region desirable from a clinical standpoint. The relation of the intraabdominal viscera to the parietal peritoneum must, however, in large part, be reserved for the study of the visceral peritoneum. A strict adherence to this plan will not be profitable, for some of the structures bear important relations both to extra- and intraperitoneal structures, so that one is constantly required to sacrifice topographic exactness for clinical experience.

The Lower Abdominal Region.—This region illustrates the concluding remarks of the preceding paragraphs. The structures here considered belong in part to the anterior abdominal region and in part to the pelvic.

The peritoneum of this region is interrupted by folds radiating from the umbilicus to the lower portion of the abdomen. Their relation is due to their embryonal origin. Because of their relation to the umbilicus, they are fittingly called the plicæ umbilicales.

The Plicæ Umbilicales.—These plicæ consist of three radiating ridges (Fig. 46). The median extends from the umbilicus to the fundus of the bladder, representing the obliterated urachus and

is called the plica umbilicalis media. Diverging from the umbilicus toward the inguinal regions are two ridges representing the two obliterated hypogastric arteries. They are called the plicæ umbilicales laterales. In some instances these ridges are prominent from the umbilicus downward. This is always the case in the new-

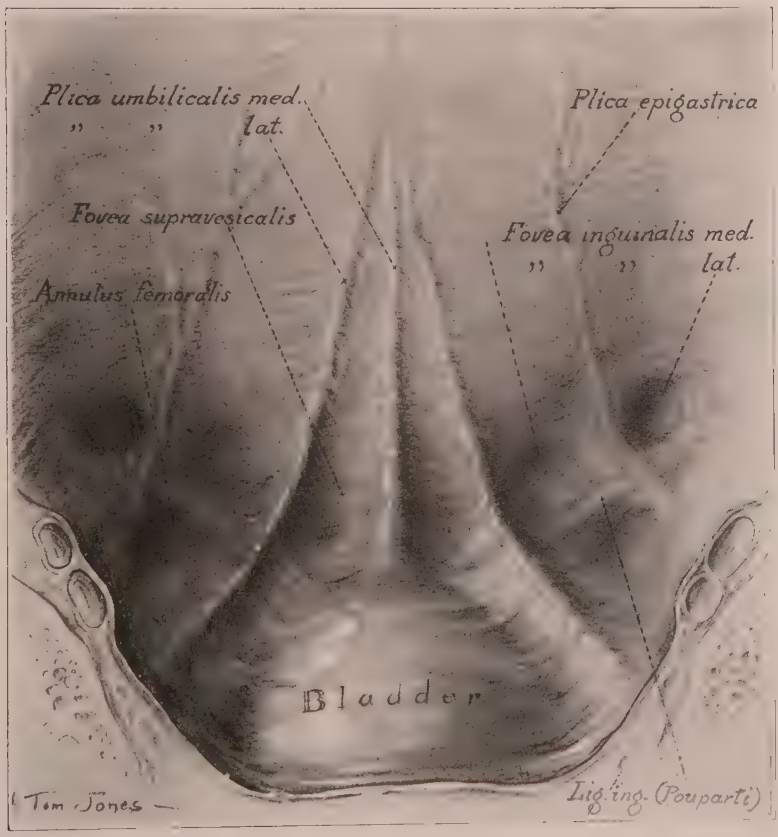

Fig. 46.—Supravesical folds and fossæ. (Redrawn from Waldeyer.)

born. In the adult the peritoneum may be closely attached to the transversalis fascia in the region of the umbilicus, and it is only when the lower portion of the abdomen is approached that the plicæ become prominent. Here it is only in the region immediately below the umbilicus that the peritoneum is free from undulations.

The Plica Epigastrica.—In addition to the plicæ already noted as due to vesical remains, the plica epigastrica, produced by the deep inferior epigastric artery off each side, is worthy of note. It occupies the position of this vessel, beginning immediately over Poupart's ligament and extending a variable distance cephalad medially. It is always easily recognized and is an important landmark of this region. It is of particular importance because it harbors the inferior epigastric artery and vein which are vessels of importance in their relation to operations on hernia.

The degree of plication produced by these structures is very variable. In many subjects, particularly in those in which there is an abundant deposit of preperitoneal fat, it may be but little marked. In all instances they are sufficiently well marked to divide off the lateral and supravesical regions into various depressions of foveæ.

The result of the presence of these plicæ is the production of foveæ which are of interest as they are the sites of exist for hernias in this region.

The Fovea Supravesicalis. This is a depression, usually but slightly marked, situated on the summit of the bladder between the median umbilical plica and the lateral umbilical plica. This depression is rarely the site of a hernia. Berger has collected a number of instances.

Fovea Inguinalis Medialis.—The fovea inguinalis medialis is situated between the lateral and the epigastric plicæ. This depression is always easily recognizable and is the site of direct inguinal hernias. It is a triangle, having the obliterated hypogastric artery medially, the epigastric artery laterally, and Poupart's ligament at its base. The peritoneum in this situation is not closely attached, so that, if the conjoined tendon is defective or becomes weakened, the abdominal contents may protrude. Inasmuch as this region does not represent a canal, a hernia in this region must be classed with hernias of the abdominal wall, and not with the inguinal hernias. In consequence the peritoneal covering of the protrusion does not assume the importance it possesses in inguinal hernias.

The Fovea Inguinalis Lateralis.—This fovea lies lateral to the epigastric plica and above Poupart's ligament. It is nearly always deep enough to admit the tip of the finger. This fovea is

of greater practical interest than either of the preceding ones, for it is at this point that the testicle escapes from the abdomen, dragging with it its adnexa and the peritoneum and forming the avenue of escape from the common inguinal hernia. At first, after the descent of the testicle, there is a tubule of peritoneum extending from the internal ring to the testicle. At the seventh month this canal usually begins to become obliterated, and usually but a slight depression remains at birth. In about a fourth of the cases, however, the canal is open at birth and is then a potential canal for hernia. During the first four years following birth some of these canals become obliterated, while a considerable portion remain open and become the site of hernias. Hessert contends that the hernial sac in all inguinal hernias is congenital.

The interesting point in this connection is the causes active in producing a late obliteration of the sac. The factors at work here are, no doubt, similar to those which have to do with the conglutination of the mesentery in other regions of the body. From the topographic relation of the peritoneum it is certain that in some manner the endothelium disappears and the fibrinous tissue becomes united. It is impossible that the sac becomes eliminated by a regression of the peritoneum which has been supposed to account for the obliteration in other situations. The means which nature employs are indicated by the points at which obliteration is earliest and firmest, at the internal and external rings. The factor operative here is the pressure of the rings themselves. After the testicle has passed, the fibrous tissue of the rings is free to compress and obliterate the sac. That pressure is important is indicated by the fact that, when the testicle is retained within the canal, very commonly the peritoneum does not close at the lateral ring. That pressure may cause obliteration of peritoneal surfaces is indicated by the facility with which two peritoneal surfaces may be made to unite when pressed together by two metal plates as demonstrated by Maximow. The use of the Murphy button illustrates the practical application of this principle. The younger the animal, the more readily such a union is brought about. Insofar as it is possible to account for peritoneal conglutination by physical means, the pressing together of opposed surfaces may account for it, and the inguinal canal furnishes the most convincing

argument in favor of this theory. This fact is of interest in connection with the question of curing hernias in children by means of constant pressure from a truss. Some years ago I searched the literature for evidence bearing on this, but none was discovered (Hertzler). From a theoretic standpoint it is questionable whether the pressure from a truss is sufficient and constant enough to exert such influence.

Even when this canal has been obliterated its site, as already noted, is represented by a dimple that usually admits the tip of the finger. The peritoneum in this region is not intimately attached to the fascia or to Poupart's ligament; and, so far as the mobility of the peritoneum is concerned, the participation of the peritoneum in the formation of hernial sacs, it is easy to understand, could take place, and it no doubt does do so in recurrent inguinal hernias.

Fovea Femoralis.—The femoral fovea does not represent a canal but merely a dimple below Poupart's ligament and medially to the vas deferens. In this area the support is weakened by the passage of the femoral vessels, and is obstructed only by a lymphatic gland and its surrounding areolar tissue. By virtue of such weakening, abdominal contents may find an avenue for escape, and a femoral hernia results. The sac is generally supposed to be produced by a sliding of the peritoneum. Sections of fetuses in this region warrant this assumption that with the lengthening of the lower limbs the larger vessels are pulled down and with them the peritoneum. In this way the peritoneum may be made to dimple as in the inguinal region. This possibility is further suggested by the fact that when there is no hernia frequently a small lipoma lies just beneath Poupart's ligament which has fibrous and vascular associations with the retroperitoneal tissue. Still more commonly, at the tip of the hernias, external to the sac, lipomatous masses are attached.

The Pelvic Region.—The pelvic region includes the peritoneum of the true pelvis. It may be made to include also the mobile portion of the peritoneum which loops from the recti muscles to the summit of the bladder. The point of deflection from the fascia of the anterior abdominal has already been discussed. Because of the great variation of the degree of distention of the bladder

the height at which it becomes attached is variable. When moderately distended it may be said to attach near its highest point (Fig. 41).

When the bladder is filled, the fundus rises, and with it the peritoneum. As already discussed, in moderate distention the proportionate elevation is marked. In excessive distention the fundus of the bladder rises above the point of attachment, and approaches the abdominal wall, producing a deep fossa between the bladder and the parietal peritoneum of the anterior abdominal wall, forming the excavatiopubovesicalis of Waldeyer.

On either side of the bladder the peritoneum dips down to near the point of insertion of the ureters. This produces two fossæ, the paravesical fossæ. These are again divided into the fossæ paravesicales anterior and posterior by a fold of the peritoneum extending across the base of the bladder, the transverse vesical fold (Fig. 47). This fold of peritoneum is a reserve one, and may become obliterated when the bladder becomes strongly distended as first pointed out by Henle.

The Peritoneum in Douglas' Pouch.—As the peritoneum descends over the posterior surface of the bladder (interrupted by the uterus in the female), it reaches its deepest point just above the prostate. It then ascends the posterior wall of the pelvis, including the rectum as it ascends. Its smooth contour is interrupted in its course by two folds extending from the base of the bladder to either side of the rectum. These are the plicæ vesicorectales. These are of special importance because they harbor the ureter and the termination of the vas deferens and the inferior vesical artery. Above this plica is the excavatioparicovesicalis and below it the excavatiorectovesicalis. Behind the bladder the peritoneum descends to within 2 cm. of the tip of the prostate, forming here a depression between the seminal vesicles. In some instances, according to Waldeyer, it may extend as far as the tip of the prostate.

Behind and below, the rectum produces a more or less irregular bulging into this space, but as the promontory of the sacrum is approached the peritoneum surrounds more and more of the circumference of the gut so that it obtains a true meson at the upper portion. At the upper border of the true pelvis the peritoneum

becomes attached closely to the endoabdominal fascia, and is continuous with the posterior parietal peritoneum.

The Peritoneum of the Uterus and Broad Ligaments.—The relation of the peritoneum and bladder in the female is not essentially different from that in the male, save that before dipping deeply

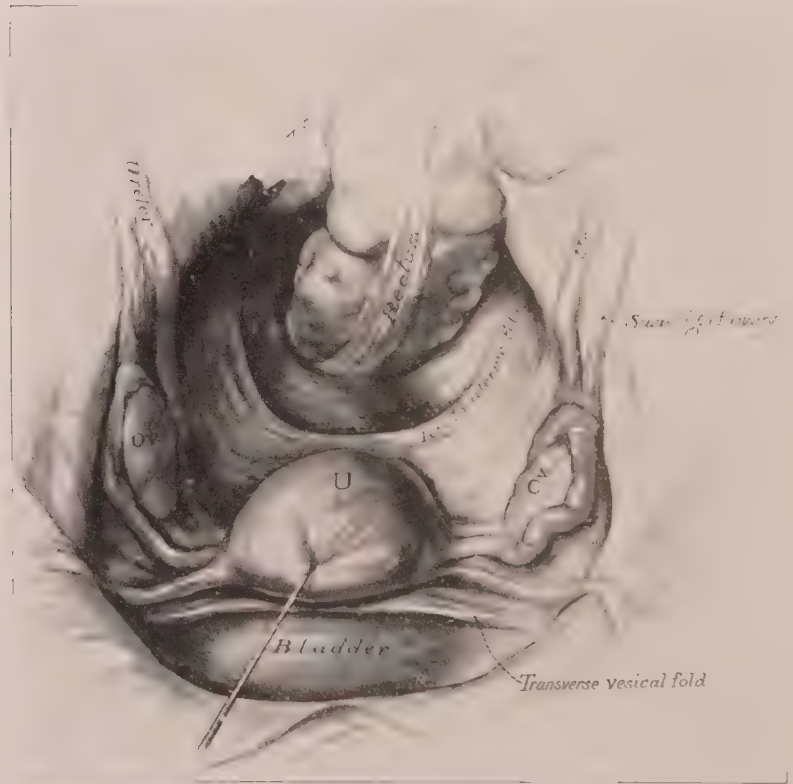

Fig. 47.—Aeroplane view of the pelvis showing the relation of the transverse vesical and rectouterine folds to the uterus.

to form Douglas' pouch it is reflected over the uterus. The line of reflection in front is about on a level with the junction of the cervix and the body of the uterus, though, for a distance of 2 or 3 cm. above, it can be readily separated.

Over the body of the uterus the peritoneum is closely attached so that even with the most careful dissection, it can be removed

but incompletely. As it descends over the posterior surface of the uterus it extends over the upper portion of the vagina, and, dipping into the floor of the pelvis to form Douglas' pouch, extends hence up the posterior wall of the pelvis. The depth to which the pouch descends is variable. Waldeyer places it as low as the level of the external os. This would seem to correspond to the majority of patients as observed at operation. Bayer shows it as terminating at the level of the internal os. My observations, made on several hundred living patients both in making abdominal and vaginal celiotomies, leads me to make the following estimate of the lower level of Douglas' pouch; in the majority of instances it reaches about 1 cm. below the level of the internal os in nonparous women. In women that have borne children it is nearer the external than the internal os. In some instances, particularly in women who have rectoceles without a corresponding and concomitant desensus of the uterus, it may extend even lower than the external os. In some instances the vault of the vagina may be pulled to the level of the external os, or the peritoneal reduplication extends between the vagina and rectum to a point below the vault of the vagina.

The fact that anatomists are disposed to place the lowest depth of the cul-de-sac at a higher point than this is due to the fact that they deal with hardened material, which is not only inelastic, but actually contracted from the action of the hardening fluid.

The depth of Douglas' pouch may be much increased by pathologic conditions. With the formation of a rectocele the peritoneum may approach the perineum, and, in some cases, protrude from the vulva, forming perineal hernias. In one instance in my experience the peritoneal pouch extended four inches beyond the vulva, and contained small intestines. In two other instances smaller pouches were observed.

The Ligamenta Lata—The Broad Ligament of the Uterus—The Mesenteries of the Uterus.—The broad ligaments (Fig. 48) are broad fibromuscular planes extending from the lateral borders of the uterus to the walls of the true pelvis. These sustentacular structures are covered by peritoneum. The bulk of these ligaments is made up of bundles of connective tissue and nonstriated muscle fibers with areolar tissue between these bands. Intermin-

gling with the fiber bands are vessels and nerves. The amount of this sustentacular tissue is abundant in the base of the ligament, and nearly absent below the tube, increasing about the tube and uteroovarian ligaments. The general direction of the lower bundle of fibers is upward and lateralward beginning at the level of the internal os, while the upper bundle is directed laterally from a point just below the cornu of the uterus. In order to emphasize the identity of these bundles and to indicate their relative posi-

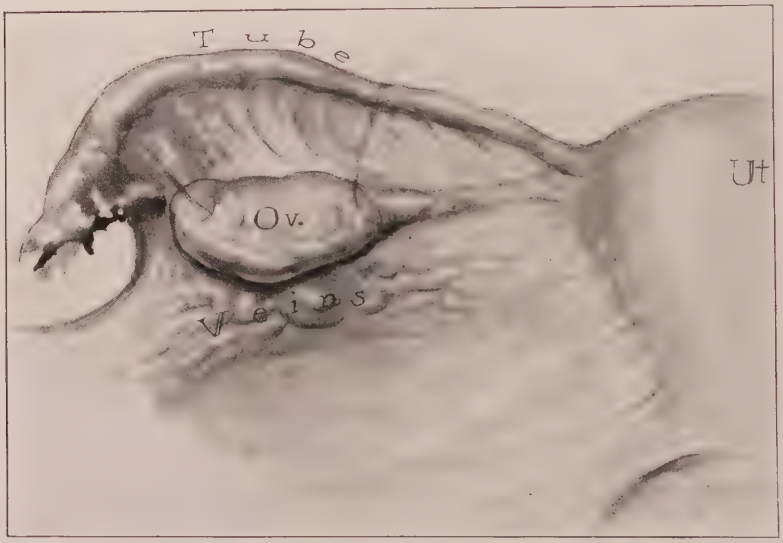

Fig. 48.—Posterior view of the broad ligaments of the uterus. Note the veins of the pampiniform plexus.

tion I have called them, respectively, the inferior and the superior uteropelvic ligaments.

The uterine ends of these bands are firmly attached to the uterus, reaching their strongest development about the level of the internal os. These fascial bands, as well as the associated muscle fibers, become less compact as the lateral wall of the pelvis is approached. They find their chief attachment about the perivascular sheaths of the great pelvic vessels. Some fibers secure attachment directly to the endopelvic fascia. In the pelvis of a young negress I found a heavy band attached directly to the fascia over the obturator muscle. In repeated dissections on other subjects no

such heavy single insertion could be demonstrated. In all cases, however, if the peritoneum is carefully dissected from the broad ligament, heavy fascial planes may be demonstrated having a direct connection with fascial structures of the pelvic wall. The dignity of these fascial planes is best appreciated when cross sections are made, and stained with some specific connective-tissue stain.

As accessory to the broad ligament is the uteroovarian ligament. It presents a strongly round band, extending from the lateral wall of the uterus to the median pole of the ovary. This is the only structure concerned with the support of the ovary worthy of the name of ligament. This band receives as a covering a fold of peritoneum, which practically forms a short meson. This fold of peritoneum extends to the hilus of the ovary but does not extend over its surface. This line of termination is known as the Farra-Todd-Waldeyer line.

The round ligaments are fibromuscular bands extending from the anterior inferior aspect of the uterine ring. In their course from the uterus to the point of exit they elevate the peritoneum somewhat, but never to the extent of forming a meson.

The structures above enumerated comprise the sustentacular elements of the uterus. Over these are draped folds of peritoneum like a sheet over a patient in an improvised vapor bath, which serves the purpose of protecting the intestines from friction against the structures beneath. The point to be emphasized is that the peritoneum covers but does not enter into their formation and it does not exercise a sustentacular function. The statement in the work on anatomy, that the broad ligaments are made up of folds of the peritoneum, is not sustained by the facts.

From the foregoing it is evident that the peritoneum must form two main folds, extending laterally from the uterus to the pelvic wall, to which are added accessory folds covering the round and uteroovarian ligaments. Between the inferior and superior uteropelvic ligaments the two layers of peritoneum come in contact. This area is transparent so that the lines in the surgeon's fingers readily show through, being as free from blood vessels as is the mesentery of the small gut. Because of this thinness and freedom

from vessels this is the point usually selected for the passage of the ligature securing the ovarian vessels lateral to the ovary.

The mesosalpinx, projecting from the mesial half of the superior border of the broad ligament, contains between the peritoneal

Fig. 49.—The genitoenteric fold made prominent by forcible traction on the terminal portion of the ileum.

folds some nonstriated muscle fibers and many small vessels which supply the tube and in the lateral part the parovarium. This meson, therefore, is not transparent.

Accessory folds radiate from the main peritoneal planes. The

best marked of these is the plica genitoenterica of Treitz. This fold extends from the lateral termination of the broad ligament and extends lateralward and upward and may reach the ileum (Fig. 49). It is essentially the plica of the ovarian vessels. Savage emphasizes the prominence of these vessels, but does not indicate their relationship to the fold now under discussion. If the appendix happens to lie in the general direction of the course of the ovarian vessels this plica extends to that structure in many instances. If the cecum is high and far out this fold may extend

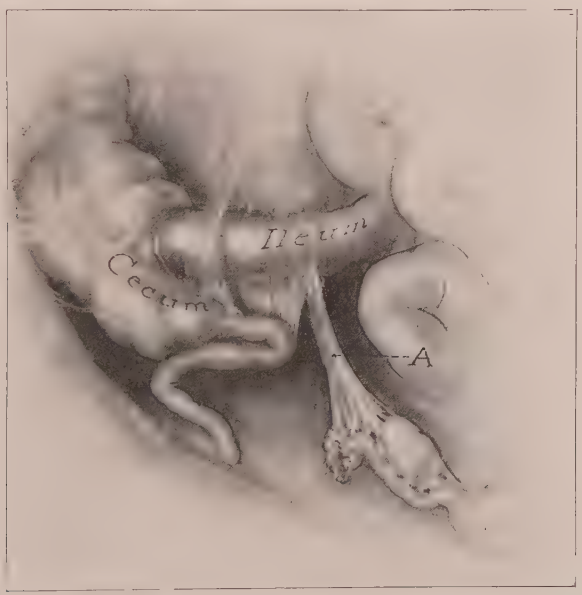

Fig. 50.—A prominent genitoenteric fold extending from the ovary to the terminal portion of the ileum. This represents the ilioovarian ligament of Durand.

from the terminal ileum to the ovary (Fig. 50). In such an event it represents the ligamentum ilioovarian of Durand. When the cecum hangs over the pelvic brim this fold may be continuous with the under surface of the mesoappendix or with the meson itself, if this arises from the posterior surface of the cecum. In such event, it exemplifies the ligamentum appendiculoovarian of Clado. It is in no sense a ligament, however, but merely indicates the line of descent of the ovaries.

Under certain conditions these folds may become more extensive. If the cecum is attached high up over the kidney, or if it possesses a meson, the genitoenteric plica may extend up near the lower pole of the kidney. This is likely to be the case especially in women who have borne children or who have been the victim of large uterine tumors.

The left side of the pelvic is possessed of a genitoenteric fold corresponding to that on the right side but it has received less attention because it is not associated with such an interesting organ as the appendix. Waldeyer has noted that this fold may extend as far as the mesosigmoid. Nagel shows this band extending well over the mesosigmoid. All these are only modifications of the plica first described by Treitz.

More recently Liepmann has described a fold on the left side which he has named the ligamentum infundibulocolicum (Fig. 51). This fold spreads up high behind the plica genitoenterica to the meson of the sigmoid. This author admits that "plica" would be a better termination than colicum except for clinical reasons. This plica extends from the tip of the broad ligament to the colon (or sigmoid) and this author suspects that it may explain pelvic infections in the virtuous virgin. Bartels was unable to discover any lymphatic connection for him. Liepmann has noticed his plica particularly in women who have borne children or who have had solid uterine tumors. The prominence of these folds is of no special significance. The plicæ genitoenterica are most prominent in the newborn (Nagel), and in the child before the ovary has descended into the pelvis. When the ovary descends these folds are smoothed out. If for some reason the pelvic organs are raised up, more prominent folds again appear. That any of these folds may be of such extent as to cause any embarrassment, as Liepmann believes, seems unlikely.

Uterosacral Ligaments.—Behind the uterus two pairs of folds of greater or less prominence may be seen. The best known of these represents the uterosacral ligaments, which extend from the lateral border of the cervix at the point of juncture between this structure and the origin of the bases of the broad ligament (Fig. 51). They cover musculofascial bands, which are believed to hold the cervix in the hollow of the sacrum. These folds begin about

the level of the internal os and disappear in the fold of the pelvic colon. They contain very few muscular fibers, which may seem to substantiate a belief in their sustentacular function, yet plication of them to restore the wandering cervix to its normal position has not met with success. The reason for this may be that

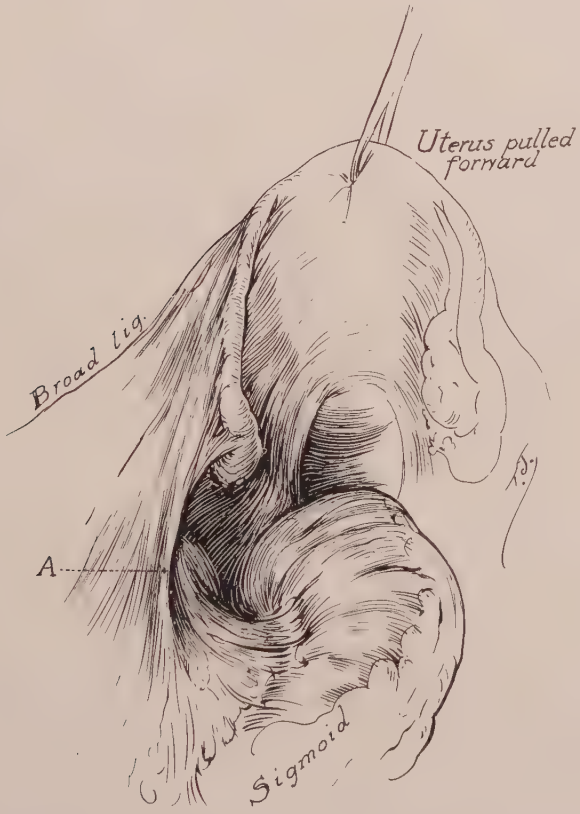

Fig. 51.—The uterus is pulled violently forward and downward to make prominent the ligamentum infundibulum (*A*) of Liepmann. At the base of the uterus the uterosacral ligaments stand out prominently.

their shortening has not been properly done. Mere coaptation of the peritoneal folds can not be expected to secure permanent results. Recently Somers and Blaisdell have studied the problem anew with the full understanding that the sustentacular substructures lie beneath, and are independent of, the peritoneum.

Uterolumbar Fold.—Above and lateral to these are less constant folds, the ligaments uterolumbalis of Vallin. These folds extend upward and outward, to be lost in the peritoneum over the lateral border of the promontory. In my experience this fold corresponds to the iliac vein. With complete fusion of the cecum and the terminal ileum this fold may be continuous with a fold reaching to the ileum. When these are present a violent pull upward on the ileum proximal to this fold will produce a "kink" which has been mistaken for a pathologic structure and, has, consequently, been the object of vehement verbal and operative attack.

It is of practical importance to note that the peritoneum covering the various ligaments and folds is continuous with the peritoneum of the adjoining parietal peritoneum. It falls in folds, as it were, over the fixed structures beneath. It is more to be regarded as a cause of traction on underlying organs than the skin over an ankylosed joint, and not more fit for fixing too mobile organs than a fold of skin over a fracture.

The Parietal Peritoneum of the Posterior Abdominal Wall.—The parietal peritoneum of the posterior abdominal wall must be thought of as the floor of the cavity in which the surgeon works. This floor covers many structures with the exact location of which he must needs be familiar. From it project organs which he opens the abdomen to attack. Each of these lies far from the source of its blood supply. The blood vessels are enclosed or covered by fibrous sheaths. The various septa divide the abdominal cavity into a series of compartments, which are often of importance in the limitation of infections or in determining in which direction infections will spread. Because of this complexity each fold and fossa which may aid in orienting the operator is worthy of record.

In order to facilitate the charting of this area the peritoneum of the posterior wall may be considered as being divided into an upper and a lower compartment by the transverse mesocolon. The upper contains the liver, stomach, pancreas and spleen; the lower contains the small intestines and the pelvic organs. This dividing membrane was called by Hyrtl "Diaphragma secundarium." Debierre expressed the relationship graphically as follows: "Deux etages adbominaux qui resultent de l'existence de mésocolon trans-

verse.'' The importance of the transverse mesocolon as a topographic landmark was more fully recognized by Waldeyer.

When the transverse colon and the great omentum are lifted up the mesocolon stands up like a partition between the two halves of the abdominal cavity (Fig. 52). Below lie the small intestines, enclosed on either side by the ascending and the descending colon. This area is further divided in the median line by the mesentery of the small intestine, which passes from above downward more or less obliquely to the right to reach the ileocecal junction.

The study of the mesenteries is of fundamental importance. The mesenteries indicate the situation of the blood vessels, and one has but to follow the presenting part to its attachment in order to determine the source of the blood supply. The determination of this is of importance because in any operation on the gut tract, or on the solid organs the source of the blood supply must be known before any procedure dare be undertaken. The inadvertent ligation of a blood vessel supplying a considerable segment is of course inexcusable bungling, but the injury of important trunks with the associated hematomas and subsequent thromboses is not so uncommon, and might be lessened by a more painstaking attention to detail.

As the peritoneum is traced upward from the pelvis, before it is interrupted by the attachment of the mesenteries of the various organs, it covers loosely the great vessels of the lower abdomen. This attachment is such that, while it is loose enough to permit access to these vessels, it is firm enough to resist the spread of infection across the midline. Lateral to the vessels, where the lymph nodes lie, the attachment is still looser, permitting ready dissection. More lateralward the peritoneum is quite free from the muscles so that the underlying structures are reached with the greatest facility. This is of importance in operations on the ureters. The unfavorable factor in this relation is that pathologic collections find little resistance in this region, which is evidenced by the fact that infections extending over the brim of the pelvis spread over the muscles as already noted in the discussion of the pelvic peritoneum.

Laterally to the colon the peritoneum passes over and envelops the kidney and receives upon it the ascending colon on the right

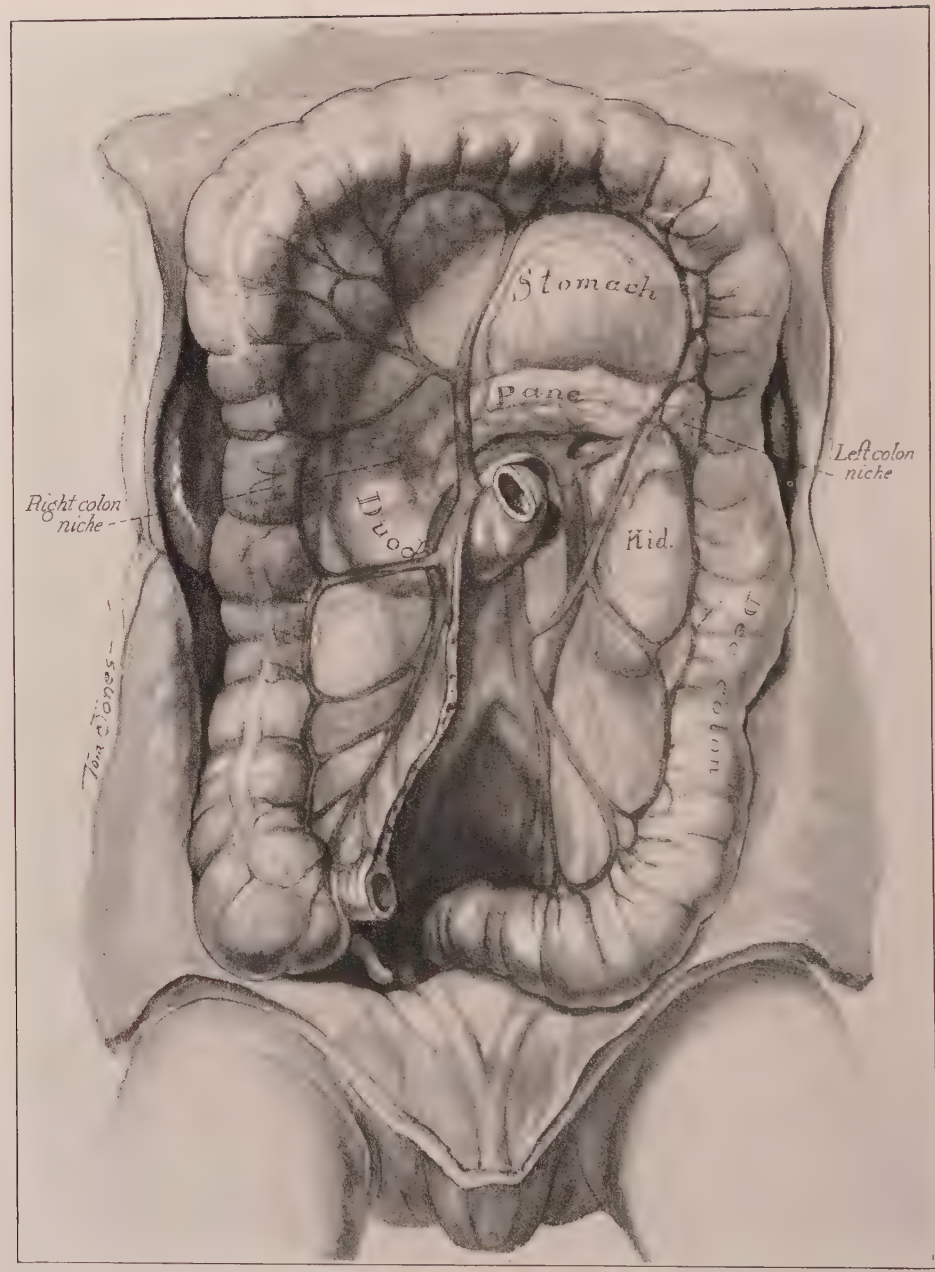

Fig. 52.—The colon and its mesentery are drawn forcibly upward, showing the relation of the attachments of the meson to the kidneys and pancreas. (Waldeyer.)

side and the descending colon on the left side. In the adult the fusion between the primitive parietal peritoneum and the peritoneum of the colon is so complete that the parietal peritoneum appears to pass over the colon, that is, the posterior surface of the colon appears actually retroperitoneal. The same thing is true of the left side. Lines of fusion are not so complete, however, but that at least some remnant remains which forms some barrier to the spread of infection. These barriers are scarcely demonstrable by sharp dissection, but are readily demonstrated by methylene blue injection. This explains why infections lateral to the colon so seldom reach the space medial to the colon. One would naturally expect a free spread in this direction since, medial to the colon, the parietal peritoneum is floored by loose areolar tissue in which are imbedded vessels and lymph glands, and finds no obvious barrier until the midline is reached, where the aorta and vena cava form a ridge from above downward. The division of the posterior parietal peritoneum by the mesocolon by the root of the mesentery does not affect the retroperitoneal space.

Medial to the colon loop the posterior abdominal wall is divided, as already noted, into lateral compartments by the attachment of the mesentery of the small intestines. This is a matter of some importance in infection in these regions, but its chief importance lies in the fact that this line gives a sure guide to any particular part of the gut, suspended by it, which we wish to reach. This will be further considered in a subsequent section. Here the chief interest centers in it as a guide to the structures lying behind the peritoneum.

As a result of this division of the abdominal cavity into two divisions by the transverse mesocolon, as above noted, numerous spaces and pockets are walled off. Henke described two triangles lying below the mesentery. These were later more fully described by Waldeyer as colon niches ("Kolon Nischen"). These, according to Waldeyer, are bounded by the transverse colon and the attached mesentery above, and on either side by the ascending and the descending colon, respectively. These two sides are separated by the bodies of the vertebræ and the root of the mesentery (Fig. 52).

On the right side the niche may be located by noting the point where the mesocolon crosses the descending portion of the duode-

num. The midline is formed by the root of the mesentery and more distinctly by the superior mesenteric vein (Fig. 52). The outer border is formed by the ascending colon. This fossa contains a part of the descending duodenum, called by Waldeyer the "pars infracolica duodeni." Laterally is the recessus renalis, which points upward and laterally. In this fossa, which barely admits a finger, the lower pole of the kidney is easily palpated; and this is the point where the ureter begins and which presents the most unobstructed site for the palpation of renal calculi. Here also, by palpating the renal vein, extensions from hypernephromata or other tumors may be palpated. Medialward, where the duodenum passes beneath the part of the mesentery, the superior mesenteric vein is encountered and the artery is found to the left of this.

On the other side of the abdomen, the pouch is bordered above again by the mesocolon, on the right by the ascending portion of the duodenal flexure, and the associated fossæ, are found. This pouch extends somewhat higher than the right owing to the ascent of the colon to the splenic flexure. At the upper part of this pouch the body of the pancreas is easily palpated, and laterally the left kidney and its vessels and ureter as in the right side. The most frequent occasion for the exploration of this region, however, comes in searching for the jejunal loop preparatory to gastroenterostomy. By swinging the finger toward the median line along the upper confines of this space, the finger is led unerringly to the desired section of gut, or comes in contact with the plica duodenojejunalis.

The intracolonic region, when all parts are in their normal position, is covered over like a watershed, by the great omentum. This in a measure shuts off this space from emanations from above, as one frequently sees in perforated gastric and duodenal ulcers.

The Supracolonic Region.—The region above the mesocolon (diaphragma secondarium of Henle) is divided into various fossæ by the organs occupying this region. This space is difficult to define. In a surgical sense it includes the lesser peritoneal cavity (the bursa omentalis), the space anterior to the stomach and its mesenteries, and the space in front of the great omentum. The importance of this broad conception is recognized when pathologic states are considered. When the stomach and gall bladder, the most trouble

some occupants of this region, infect their environment, either the lesser peritoneal cavity becomes involved or infection extends anteriorly downward and laterally over the colon. Conversely, infections arising from below, as from the appendix, tend to ascend lateral to the colon to reach the hepatic regions. Since the life of the patient so often hangs on the ability of the surgeon to go directly to the seat of the abscess, it follows that the most exact knowledge of the confines of such abscesses is indispensable.

It may be profitable to review the general territorial boundaries of this space. Above, that is, cephalad to, the attachment of the transverse mesocolon the peritoneum covers the pancreas, the suprarenal glands, and the large ganglia. It ascends to the diaphragm, forming the posterior wall of the lesser peritoneal cavity being interrupted by the various attachments of the hepatic ligaments. Escaping toward the anterior portion of the diaphragm, it finds a more intimate attachment along the central tendon. Still more anteriorly at the muscular portion of the diaphragm it is again more loosely attached. It is deflected downward to cover the underlying surface of the liver to the lesser omentum, behind the stomach, to reach the colon as the posterior layer of the gastrocolic mesentery.

The anterior layer starts above the colon, ascends to the stomach, and follows the lesser omentum to the liver, which it covers, to reach the diaphragm, which it follows to the anterior abdominal wall.

After the anterior abdominal wall is reached the attachment is looser because of the greater amount of abdominal fat in this region; and at the sites of the round ligament of the liver it forms a loose fold, comparable to that in the broad ligament of the uterus. In the region of the spleen it forms a fold to give support to that organ and then ascends to the diaphragm.

The diaphragmatic peritoneum has been the subject of much discussion, due to the fact that the tendinous portion has been a favored object for the study of minute anatomy of the peritoneum, which, in turn, has been brought into prominence in the discussion of absorption from the peritoneal cavity. This prominence is not deserved, as has already been set forth in the chapters devoted to the histology and the physiology of the peritoneum. Over the mus-

cular, as well as over the tendinous, part of the diaphragm the peritoneum is closely attached so that it is possible to remove it but incompletely, even by sharp dissection. The sparseness of the subperitoneal connective tissue in this region limits the capacity of the peritoneum to respond to infections. Because of this it is more vulnerable than in many other regions.

The Gastrohepatic Region—The Lesser Peritoneal Cavity (Fig. 53).—This region has already been briefly described as being lined above by the under surface of the liver, anteriorly by the lesser omentum, below by the mesentery of the transverse colon, and behind by the posterior parietal peritoneum, thus forming a pocket completely closed save for the opening below the hepatoduodenal ligament, the foramen of Winslow. The confines of this space may now be considered in detail.

In front the lesser omentum is attached to the liver along the margin of the portal fissure and to the bottom of the fissure of the ductus venosus. Below, it is attached to the lesser curvature of the stomach and to the duodenum for 2 or 3 cm. lateral to the pylorus. It is composed of two layers which, on the right margin, take the hepatic vessels and ducts between their folds. To the left it attaches to the esophagus and to the diaphragm and between the esophagus and the opening for the vena cava.

The gastrocolic omentum, the mesentery of the transverse colon, forms the anterior wall of this space below the stomach. It is attached to the greater curvature of the stomach above, and to the transverse colon below.

To the left this omentum extends from the fundus of the stomach to the hilus of the spleen, and forms the anterior layer of the mesentery of the spleen. This forms the lateral wall of this space, and separates it from the hepatophrenic space.

The space under discussion well deserves the appellation "cavity" for it is excluded from the greater peritoneal cavity save at the slit-like opening, the foramen of Winslow. This opening is bounded anteriorly by the hepatoduodenal ligament, below by the upper surface of the pylorus, above by the quadrate lobe of the liver, and behind by the parietal peritoneum. It derives its chief importance, in a surgical sense, from the fact that the pancreas lies in the floor of it, and when this organ becomes diseased

its space is encroached upon, as from a cyst, or exudations occur in it as in necrosis of that organ.

As a result of reactive processes this opening may become closed, converting the lesser peritoneal cavity into a closed sac. In that event, if accumulation occurs in this space, a veritable cyst may result. I once observed a patient whose foramen of Winslow be-

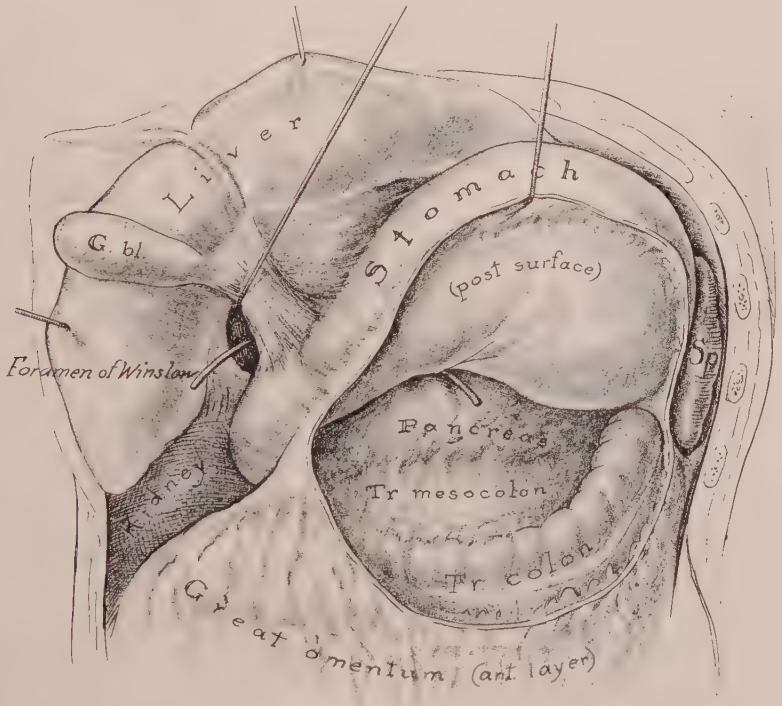

Fig. 53.—The gastrocolic mesentery is severed and the stomach lifted up, showing the relation of the pancreas to this space and to the colon. A probe is directed through the foramen of Winslow.

came occluded following drainage for acute pancreatitis. A cyst the size of an adult's head formed and elevated the liver and depressed the stomach.

The Subhepatic Region.—This region may be made arbitrarily to include the terminal portion of the ascending colon, the hepatic flexure, and the beginning of the transverse colon. To this may be added the under surface of the liver lateral to the hepatic duct.

This space has the suprarenal gland and the upper pole of the kidney behind it. Its chief claim to distinction, however, is due to the fact that it has such famous trouble-makers as the gall bladder and duodenum at its mesial-anterior border.

The sins of this region are many, but it suffers unduly because of its reputation. Developmental bands, purposeful and useful, are often seized upon as evidence of past dereliction. As has already been noted in the chapter on development, as early as the second month the colon has become fused with the parietal peritoneum over the duodenum. The cecum lies in the direct line of the transverse colon or pointed a little downward. It lies close under the liver, and does not possess a mesentery, contrary to the schematic drawings of the books. The great omentum is already attached to the colon in the region of the pylorus. The colon at this stage is smaller in diameter than the jejunum, which lies below it. The rapid development of the colon and the proportionate regression of the liver produce traction lines which not infrequently present as definite folds.

The hepatic flexure of the colon extends upward to a varying degree. Usually it forms an arc of a circle over or medial to the suprarenal gland. Sometimes the loop extends further upward and outward, resembling then the splenic flexure. The result, then, is that the terminal portion of the ascending and the beginning of the transverse colon lie more or less parallel. This arrangement has awakened memories of the fowling pieces of their youth in imaginative observers, and they have designated it "the double-barreled colon." In these cases not infrequently there is a prominent band extending lateralward and upward from the flexure to the diaphragm much resembling the phrenocolic ligament on the left side. If the transverse colon is very long and, in consequence, loops toward the sacral promontory the likeness to the splenic colon is enhanced. There is no evidence that this condition has pathologic significance; the colon is longer than the average, and, like the tall traveler in a pullman berth, does the obvious thing, to apply the simile above quoted to the latter, one might speak of the "double-barreled" traveler.

Since the hepatic angle of the colon is situated just lateral to the descending limb of the duodenum, as the gut pouches outward,

traction lines are formed, extending from the point of fusion to the point of final location. Several of these have received special names, the "hepatorenal" and the "duodenorenal" (Fig. 54). It is really the diastasis of the liver and colon, and not the kidney, that is concerned in their formation. They should, therefore, be called the hepatocolonic and the duodenocolonic folds.

Between the ascending portion of the colon and the parietal wall, also, irregular bands are sometimes noted, similar to those

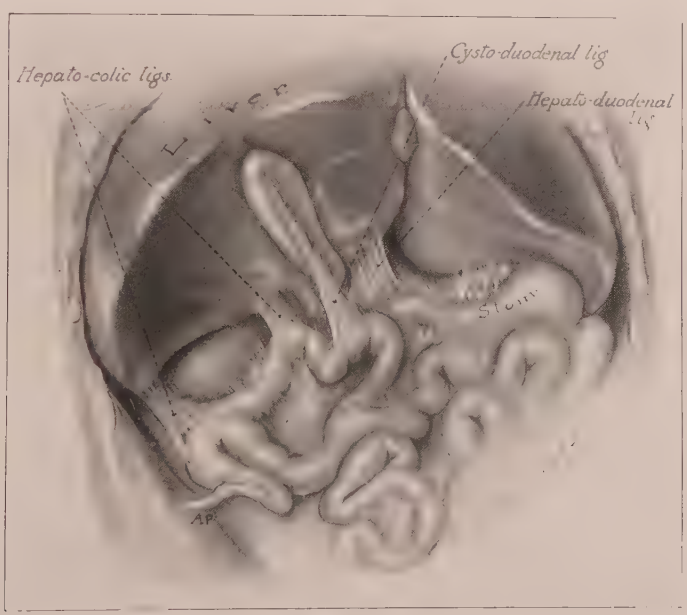

Fig. 54.—The various ligaments extending from the liver to the hollow viscera are well marked. The hepatoduodenal ligament is shown extending over the duodenum to the colon. (Seven-month fetus.)

noted in the region of the cecum. These are particularly prominent in the fetus, though they are often persistent to a lesser degree (Fig. 55). Flint has called attention to these, and had figured many examples, one of which is here reproduced. They are more often present than absent in the fetus.

Of like character, but more striking, is the falciform band, extending from the duodenum to the gall bladder to well over the duodenum. When the viscera lie in place this exerts no traction,

and in nowise hinders the movements of any of the organs. It is only when the liver is lifted sharply upward that it is put on tension, and seems to pull on the guts below. It is not abnormal, is not the result of any pathologic process, causes no symptoms, and does not require removal by the surgeon. Its only obvious use is for purposes of demonstration to anxious friends when an innocent gall bladder is cut down upon.

Toward the median line the colon receives a complete meson,

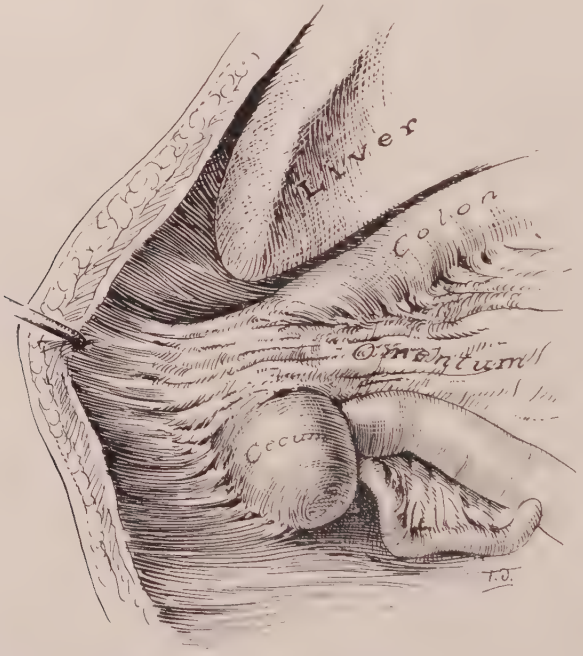

Fig. 55.—The omentum extends over the ascending colon to the parietal wall forming a parietocolic ligament. (Redrawn from Flint.)

so that the transverse colon may be elevated completely from the underlying structures. This meson is the product of the primary mesentery of the colon and of the great omentum as it advances from the median line lateralward. Beginning at the colon the upper layer of the mesocolon, which defines the lower border of the lesser peritoneal cavity, may be followed upward to its attachment with the posterior peritoneum. This attachment usually takes

place over the body of the pancreas. From this point it passes over the suprarenals and great ganglia to reach the diaphragm. At the left it is continuous with the spleen, forming the posterior leaf of the gastrosplenic ligament (better called mesentery). At the right it is continuous with the peritoneum of the subhepatic region.

When the hepatic angle is sharp and the loop long, the right border of the great omentum may reach across the two limbs of the loop. Sometimes the hepatoduodenal ligament may extend over both portions of the colon. This is most likely to be pronounced when the transverse colon is very long, and, in consequence, makes a long excursion downward toward the sacral promontory. This arrangement is of utter indifference to the patient, and is at most an esthetic offense to the artistic observer. The hepatoduodenal ligament above mentioned, extending from the liver to the duodenum, may be very prominent, especially when extensively reinforced by the mesenteric root where the duodenum curves around it. The duodenocolic ligament is formed by the early attachment of the primitive colon to the first part of the duodenum. This adhesion occurs when the great omentum is yet a small pouch. As the omentum develops it becomes attached to the colon, first, at the region of the pylorus, and then extends for a variable distance to the right side, and may add materially to the complexity of this ligament.

These bands have no other significance than to indicate the extent and direction of the excursion the large gut has made in relation to the fixed structures about it. They are phylogenetic in significance, and bear no relation to infective processes. When their substance harbors the product of inflammation they increase greatly in prominence, and may simulate adventitious bands. Even then they do not require section. If the diseased condition is conquered they recede to their former dimensions.

On the right between the liver and the colon, lateral to the hepatoduodenal ligament is a shallow fossa. In its depth lie the upper pole of the right kidney and the suprarenal capsule. Morris has described the fossa on the right side, and it is known in this country as "Morris' pouch." This pouch is often the site of abscesses, both from an ascending infection from the appendix and

from the perforation of the gall bladder, or from a gut ulcer in this region. It is worth noting that fluid in this fossa when it escapes meets the mesocolon below from the lower border of which the great omentum spreads over the small intestine. The result of this is that fluid escaping from this fossa glides over the membrane to the region of the cecum or pelvis. In the corresponding region on the left side Hildebrand described a fossa. It is covered by the

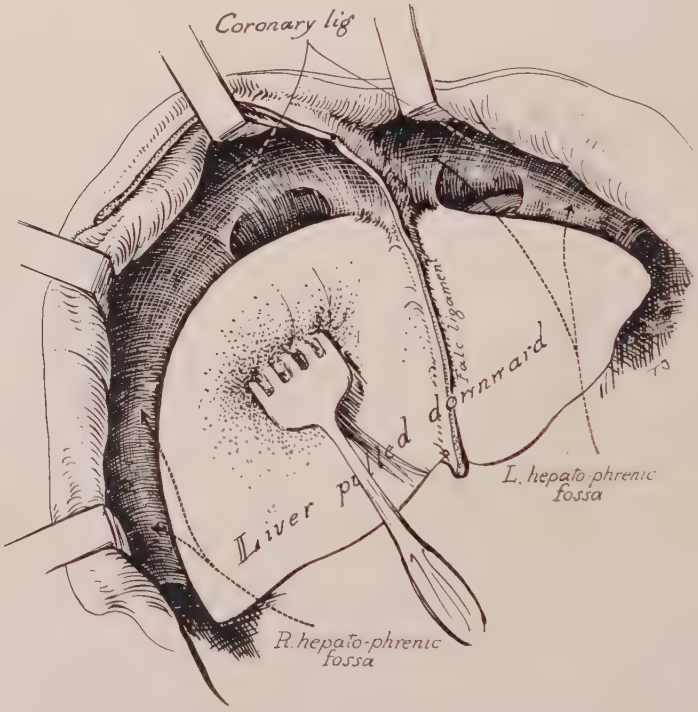

Fig. 56.—The liver is drawn downward and the diaphragm upward exaggerating the phrenohepatic space. (Semidiagrammatic.)

fundus of the stomach, and is bordered laterally by the spleen. It is a part of the lesser peritoneal cavity.

The Phrenohepatic Region.—Following upward from the renal region the peritoneum extends over the under surface of the diaphragm up as far as the coronary ligament and to the median line as far as the falciform ligament. These ligaments form a pocket lying between the liver and diaphragm (Fig. 56). It is open to the

right, and continuous with the space lateral to the right kidney and the ascending colon. This fact makes this space of importance in infections in the region of the cecum. Not only do these confines form a space within the peritoneal cavity, but the close relation of these ligaments to the endoabdominal fascia makes a certain degree of limitation in the infections of the retroperitoneal connective tissue.

The nature of the peritoneum of this region makes it of peculiar significance. The peritoneum here is devoid of subperitoneal connective tissue and vessels. Irritation here, in consequence, produces adhesions much less readily than in other regions; therefore, when infection once reaches this space, it tends to spread over the entire region limited only by the ligaments above mentioned. When drainage is required it is important that it be placed deeply enough to insure efficient drainage of this entire region. Because of the limited power of reaction against infection, drainage is required for a longer time than in other localized peritoneal infections.

To the left of the falciform ligament likewise is a pocket corresponding with that on the right just discussed. This space is but rarely the site of abscess. For practical reasons this space is best considered in conjunction with the splenic region.

The Gastrophrenosplenic Region.—Between the terminal portion of the left lobe of the liver, the diaphragm and the stomach a fossa is found which is even more definitely defined than the analogous region on the right side (Fig. 57). Skirting along the left lateral abdominal wall over the spleen the peritoneum extends uninterruptedly over the under surface of the diaphragm as far to the right as the falciform ligament of the liver. Returning to the left over the lesser lobe of the liver, the space is limited behind by the continuation of the coronary ligament of the liver. Below, this space finally is limited by the phrenocolic ligament, which extends from the splenic flexure of the colon to the diaphragm.

The extent to which the peritoneum is attached to the spleen varies. In some cases it is along a narrow line only at the hilus, and in some instances it is attached to the mesial surface of the spleen for some distance behind the hilus. The relationship be-

tween the spleen and its peritoneum is a matter of importance in splenectomy.

The Visceral Peritoneum.—The organs which possess any considerable degree of mobility or motility are nearly completely surrounded by peritoneum. Naturally, this part of the peritoneum is most suitably designated the visceral peritoneum, though there

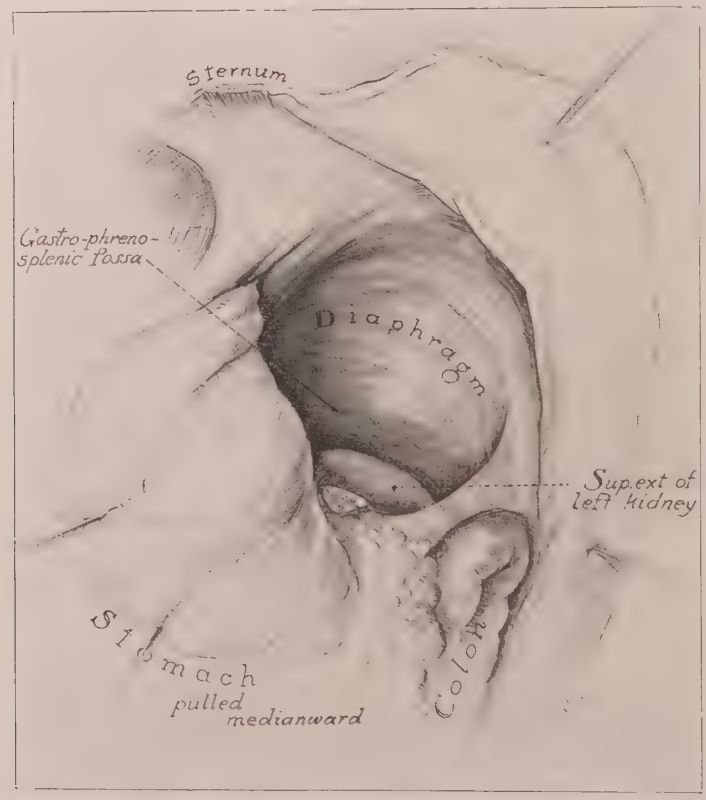

Fig. 57.—The gastrophrenosplenic region emphasized by pulling the fundus of the stomach toward the median line.

is merely a topographic and not a structural difference. The degree of completeness of envelopment by the peritoneum is, in some instances, constant, as in the case of the small intestines; while in others the degree is variable, as in the case of the vertical portions of the large gut. Organs which are not required to undergo

contraction and expansion in the performance of their own functions may be required, like the liver and the spleen, to move *in toto* to accommodate the movements of neighboring organs. Were it not that these organs are required to follow more or less the excursions of the diaphragm, the extent and complexity of the peritoneal surface might have been much reduced by placing them, like the kidney, extraperitoneally.

The mechanism by which parts of the intestinal tract reach their final position has been discussed in the chapter on development. The adult relation is but the final step in this process. It is only by following the various stages in development that a clear understanding is obtained of the relation of the various segments as they are observed in the stages of full development. By reversing the process of development the surgeon may again convert certain segments of gut into intraperitoneal organs, as it were, in order to facilitate operations upon them or to effect their removal. The duodenum and the cecum may be cited as examples of such organs.

The Arrangement of the Coils of the Small Intestine.—The small gut is maintained in position by its mesentery. The proximal width is about 10 inches, while the intestinal border is many feet in length. The mesentery must, therefore, lie in folds at its distal extremity; and for this reason it has been likened to a fan. Because of the irregularity the simile is more apt if a rural damsel's fan the day after the picnic is specified (Fig. 58). The duodenum is the only segment of the small gut not possessed of a meson, except in rare instances. Ordinarily the duodenum from its beginning at the pylorus is a retroperitoneal organ, made so in part by the passage over it of the primitive mesocolon and in part by the fusion of the gut wall with the posterior parietal wall. The duodenum is crossed by the mesocolon at about the junction of its midline and lower third. With the revolution of the colon the mesentery of the small intestine becomes twisted upon itself, forming the root of the mesentery, which still further submerges the duodenum. In rare instances fusion of the gut wall and parietes does not take place. That portion lying between the hepatoduodenal ligament and the second portion of the descending limb, retains its meson and consequently remains an intraperitoneal organ. This primitive state

is reproduced when the lower portion of the common duct is attached from behind.

The problem of depositing thirty-odd feet of small gut in the

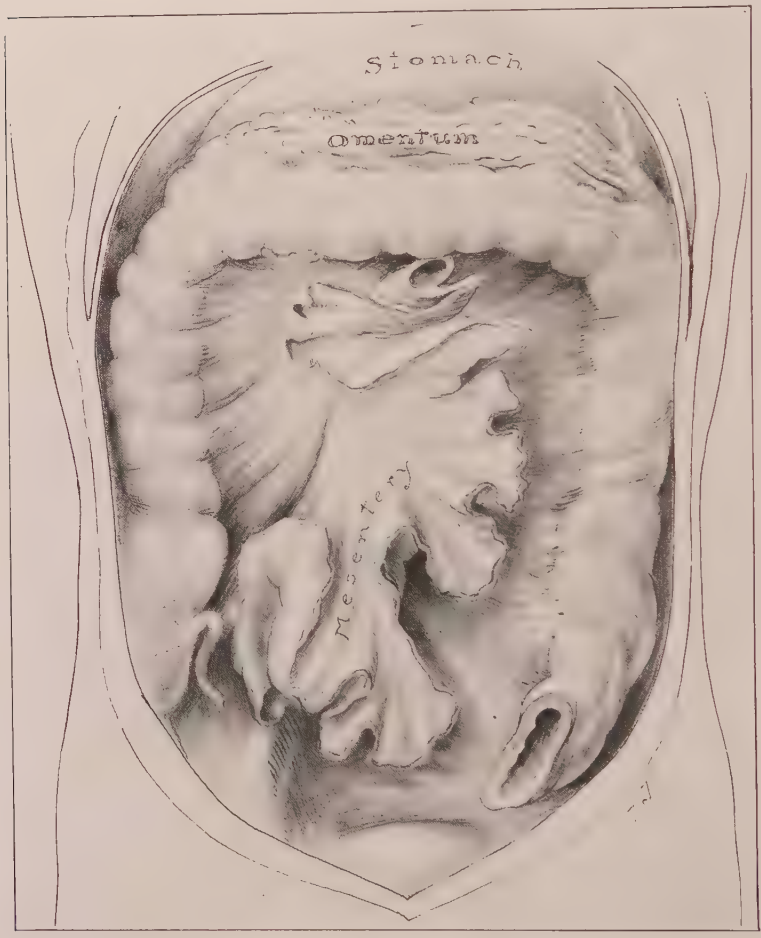

Fig. 58.—The stomach, omentum and transverse colon are raised up to emphasize the extent and fan-like arrangement of the mesentery of the small intestine. (Redrawn from Meckel.)

constricted space available is solved in an astonishingly efficient and uniform manner. True, certain variations in arrangement are frequently observed, but these are slight and unimportant. The surgeon if possessed of a knowledge of the general arrangement

can identify a loop by topography alone, with a high degree of precision.

In order to determine the topography of the small intestines a number of exact methods, to be mentioned below, have been employed by several investigators. In my studies the review in a rather superficial way, of a large number of bodies as near the normal as possible, was undertaken. In this way 1,187 cadavers hardened according to the usual method for dissecting room were studied as a basis. A smaller number of fresh cadavers were similarly examined. As opportunity has offered I have noted the topography as presented by the living patient. I was particularly interested in the arrangement of the coils, as relates to the time and the degree of descent of the cecum, and the arrangement of the colon. The unusual deviations found were compared with the clinical history.

As a result of these studies I found that in 75 per cent of the bodies there was a definite and uniform arrangement of the small gut, which may be described as follows (Fig. 59): from the jejunum a short loop extends to the left of the duodenojejunal angle and then proceeds to the midline. An additional loop may form in this region. Next there is a long loop which extends to near the hepatic flexure of the colon. It then passes directly to the left side, and forms a series of convolutions on the left side of the abdominal cavity extending to near the promontory. Thence it ascends to near the hepatic flexure and forms a descending series of loops until the promontory is again reached, when it descends into the pelvis to form a series of loops, to end by passing upward and lateralward to reach the ileocecal junction.

The second most frequent arrangement consists in a more complicated coiling of the gut in the subhepatic region (Fig. 60), then passing over the splenic region filling the left half of the abdomen, as already described, ascending in the midline to fill the lower part of the right side of the abdomen, then descending to the true pelvis, only to ascend again to terminate in the ileocecal region, as already described.

In still another series, several coils are found in the splenic region (Fig. 61), then across to the hepatic region for a few coils, then downward and to the left to fill the lower left abdominal

region and then across to complete the right side, subsequently finishing as before.

In a smaller number of cases no definite arrangement could be determined. Each case seemed to follow a different plan of arrangement. In one of the cases mentioned by Mall, there was no jejunal loop on the right side. He suggests that this absence may be due to a large colon. He suggests the loop may have been shaken out. The most of the variations in fact may be grouped about the

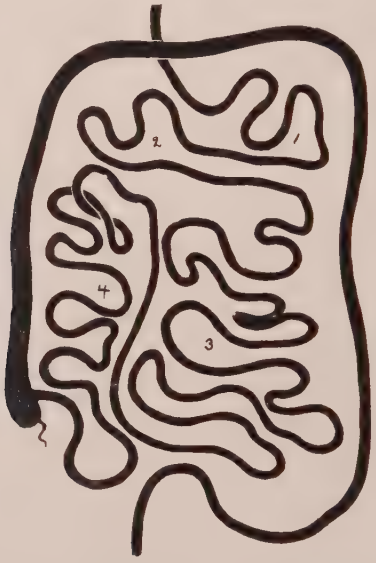

Fig. 59.—The most common arrangement of the intestinal loops. There is first (*1*) a short jejunal loop to the left of the midline, then (*2*) a loop to the hepatic flexure, then (*3*) a series of loops to the lower left, and lastly (*4*) a loop to the lower right quadrant.

variables in the right hypochondrium, whether occupied by initial jejunal or terminal ileum.

A more exact study of the arrangement of the small intestine has been made by a number of anatomists. Thus Sérnoff injected cadavers with chromic acid in order to secure firm fixation after which he made a cast of them *in situ*. In order that surface loops could later be identified he touched them with a solution of fuchsin. He studied three cadavers only, so that interest in his work is confined to his technic. Weinberg used Sérnoff's method

in the study of ten stillborn children. According to these studies in seven cases the upper segment of the jejunum lay in the upper left quadrant with the loops arranged transversely and anteroposteriorly. Following this there were irregular convolutions in the left iliac fossa. Then the midline was crossed to the right side, where the loops are arranged perpendicularly. Both these authors agree that about one-third of the whole length comes in contact with the anterior abdominal wall. Mall presents a very full and

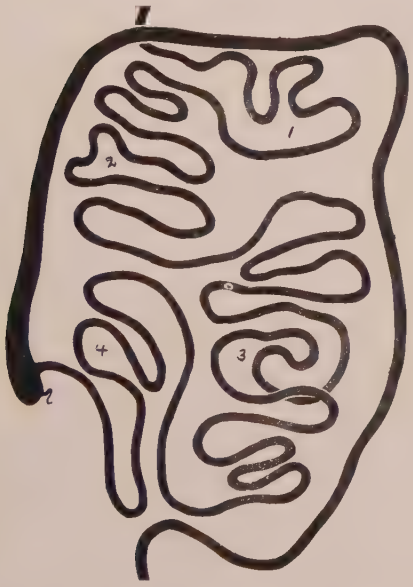

Fig. 60.—The jejunal loop (*1*) is simple while the hepatic group (*2*) is more complicated, as is that of the lower left (*3*) quadrant. The lower right quadrant (*4*) is relatively simple.

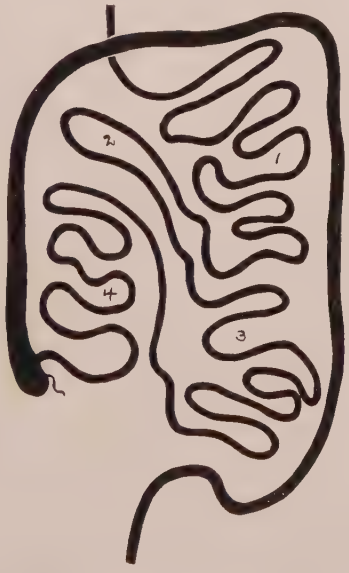

Fig. 61.—The jejunal loop (*1*) is complicated while the hepatic region (*2*) is represented by a simple loop. The left lower quadrant (*3*) is simple while the right lower quadrant (*4*) is relatively complicated.

minute discussion. He studied the problem embryologically and in 41 cadavers of adults. He accounts for the regularity of the arrangement in his series by the fact that the cadavers had been carried for several kilometers over the rough cobblestones of Baltimore's streets. Those who have ridden over the streets of this city will readily agree that, having withstood this, it may be granted that the position is the most dependent one. In addition he fixed them in carbolic-acid solution, and afterward froze them. He

notes that the general arrangement of the intestines is diagonally from the left hypochondrium to the right iliac fossa. In 21 of his cases he found the loops arranged according to a general plan. He found two loops in the left hypochondrium, and then passing to the right side at the level of the umbilicus where it forms several loops and then descends to the left iliac fossa, hence to the pelvis.

Mall's comparison of the arrangement in the fetus and the newborn with the adult is very instructive. In the fetus he found, as is also shown by Weinberg, the terminal ideal loops gradually proceed downward and with a shifting of the jejunal loops toward the subhepatic region. With this the midloops come to occupy the left iliac fossa. As the pelvis becomes developed there is a further descensus of the terminal ilial loop.

I can readily agree with this explanation, for when there is a failure of the cecum to descend there is also a failure of the jejunum to place a loop on the right side. If there is a late descent of the cecum, as indicated by oblique parietocolic bands, the embryonal arrangement of the loops may also be maintained. Conversely, in a cecum mobile the terminal loops will be found in the pelvis and a corresponding preponderance of jejunal loops in the right hypochondrium.

The most constant loop is the first portion of the jejunum, which always loops to the left. This is of interest because it is this loop that is sought for in making a junction of the gut with the stomach. The terminal ileum is much less constant, being controlled by the time and degree of descent of both the cecum and the genital glands.

When displaced the intestinal loops seek their former site unless hindered by adhesions. This can readily be verified by attaching a leaden shot to a loop, and then displacing it. Its return to the former position may be observed by the x-ray. The migration often takes place, however, before the progress can be observed by the x-ray. This is fortunate, for after extensive explorations on the intestinal tract it would be exceedingly perplexing if the internal loops should require a careful replacement.

Normal displacements occur with a general shifting of the abdominal viscera. In marked ptosis a large portion of the intestines may lie in the pelvis, as against an average of 18 per cent as

estimated by Henke. In an elongated U- or V-shaped colon the proportion of small intestines in the pelvis is usually, but not universally, increased.

Localization of Isolated Intestinal Loops.—Regular as the arrangement of the intestinal coils is, as indicated in the preceding paragraphs, it is not sufficiently so to meet the requirements of clinicians, since they must recognize the segment of gut through a relatively small incision. Other methods, therefore, have been sought to supplement the information which topography alone gives. Furthermore, the requirements of the surgeon demand an ability to determine approximately the distance from either termination of the gut and to determine in a given loop which direction one must travel to reach the terminal portion of the gut.

From the practical standpoint several sources of error appear which do not trouble the anatomists. The surgeon is concerned with the concrete case; the law of averages has for him only a general interest. This applies to both the location of the gut and its length. Besides this he has to deal with pathologic conditions. Distention of any of the hollow organs disarranges the normal; and new growths may negate any general rule.

Starting from the point where the anatomists leave the problem, we may by drawing a line from the duodenal termination to the ileocecal valve construct in a general way compartments in which certain loops will likely be found. Monks draws lines at right angles to this line forming three zones in which the first, second and third portions of the gut will likely be found. Better, in my experience, is a line drawn from the tip of the tenth rib on the right side to the tubercle on the crest of the ileum of the opposite side (Fig. 62). The first portion of the gut will most certainly be found above this line. Another line drawn from the umbilicus to the pubis will, with the terminal half of the line just mentioned, include the second portion. The space to the right of the perpendicular line (Fig. 62) will include the third portion. Such a division serves the useful purpose in that when an intestinal fistula occurs above the primary line it is probably so close to the stomach that inanition will likely take place unless it is closed, while a fistula in the third space is most certainly so far from the stomach that inanition will not follow expectant treatment. Furthermore,

fistulas in the first portion owing to the presence of digestive fluids likely will not close spontaneously within an extreme limit of from six to nine months. In but few cases are the intestinal groupings so atypical as to vitiate this general rule.

In determining the topography of the individual loops, the arrangement of the vessels in the mesentery, the presence of the

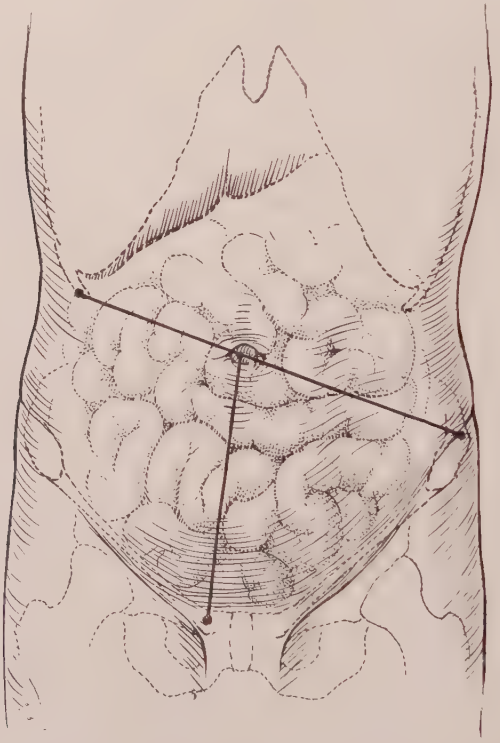

Fig. 62.—Lines drawn from the tenth rib on the right side to the left iliac tubercle. A second line from the umbilicus to the pubic eminence. The lines roughly separate off the various portions of the small intestines. The intestines above the line represent the first and second portions. These below and to the left the third portion while those to the right below represent the terminal portions.

folds of the mucosa, the valvulæ conniventes, and the presence of Peyer's patches, furnish the most reliable guides.

Monks has visualized the vascular arrangement in a satisfactory way. In conformity with the physiologic function the upper portion of the gut has the largest vessels which have the most direct

course (Fig. 63). Further down, the course of the vessels is interrupted by anastomosing loops (Fig. 64), and, as the terminal portion is approached, secondary and tertiary loops are found (Figs. 65 and 66). In addition to the absence of the accessory loops in the upper portion, the vessels have a business-like direct-

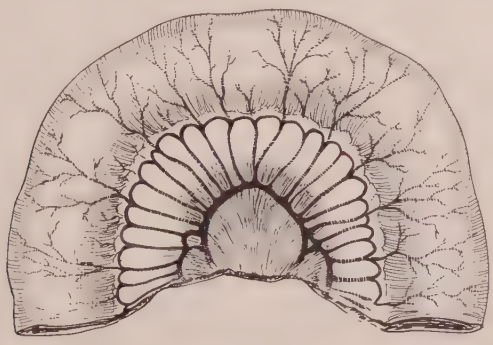

Fig. 63.—Arrangements of the mesenteric vessels of the beginning of the jejunum. (Monks.)

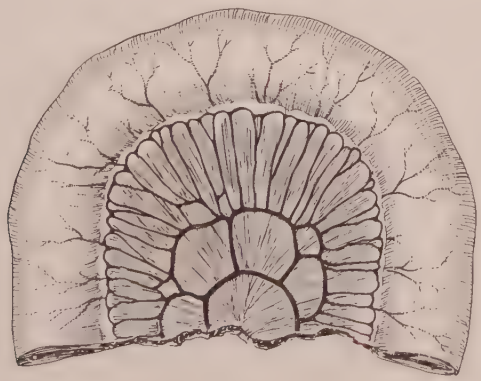

Fig. 64.—Arrangements of the mesenteric vessels near the termination of the jejunum. (Monks.)

ness, which bespeaks the provisioning of an active field of physiologic operation. As the terminal portion is approached, the vessels, in addition to the loops, show a wary course suggesting the soup- and bread-line of the slackers. In addition the vessels here are obscured by a more abundant deposit of fat (Fig. 67). This variation in vascular arrangement corresponds to definite segments

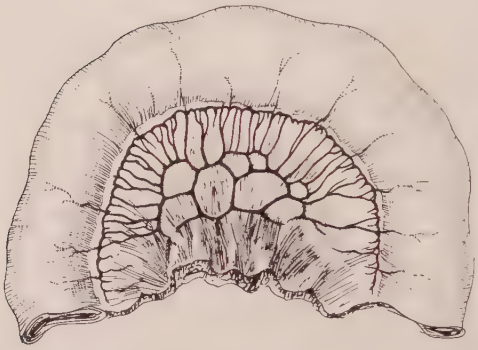

Fig. 65.—Arrangement of the mesenteric vessels in the first part of the ileum. (Monks.)

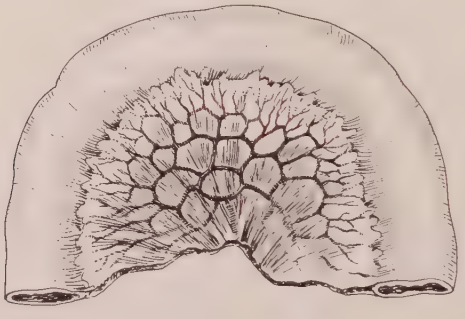

Fig. 66.—Mesenteric vessels in the midileal portion. (Monks.)

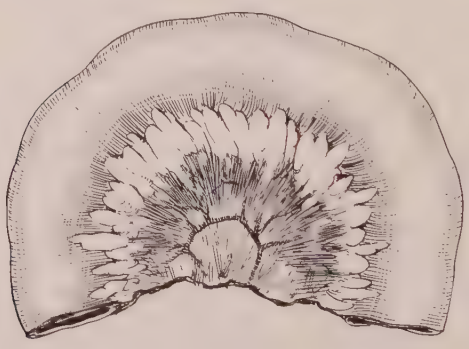

Fig. 67.—Vessels in the region of the terminal ileum. (Monks.)

of gut. Instead of expressing the probable distance in feet, as Monks does, owing to the variability in the length of the gut, segments of an arc had better be conceived rather than units of

distance. Each of the above figures represents the vascular arrangements of a certain segment. The prominence of the vessels in the gut wall is as important as those of mesentery, as also pointed out by Monks. As additional loops are interposed in the course of the vessels in the mesentery, the vessels in the coats of the gut are visible to a less degree. In Fig. 66 the number of loops largely increase, and their course becomes irregular, branching and indirect, while no vessels are seen within the wall of the gut under normal conditions. When there is much fat in the mesentery the vessels in the gut wall are usually indistinct, becoming more so as the fat increases. In judging the degree of vascularity of the gut wall the general vascular state of all the abdominal viscera must be taken into account, and the general adiposity of the individual must be noted before adjudicating the amount of fat between the loops.

Structures within the gut wall may be discernible, and give direct evidence of the segment of gut in hand. In the upper segment the valvula conniventes can be appreciated by the sense of touch. These occupy the upper third of the gut. Their entire absence indicates a more terminal portion of the gut, away from the chief field of functional activity. The presence of Peyer's patches indicates the terminal portion. In the normal gut these may be visible with difficulty. When irritated they are easily recognized. Here also palpation may aid the sense of sight. When a portion of gut is exposed, as in a fistula, the mucosa may often be inspected directly or with the aid of an air-dilating cystoscope, as in the study of fistula, and the structures above noted be identified.

The character of the intestinal contents may give evidence of the site of a fistula when one exists.

By pulling on the loop and noting the point of attachment of its mesentery, valuable topographic information may be gained. Following the mesentery with the finger to the point of insertion may aid. The relation of this point to the renal artery may help in some instances. When the radix extends high above the artery the first portion of the gut is in hand; when low, the terminal portion is being inspected.

One of the most perplexing problems the surgeon has to deal

with is to determine what direction one has to travel to reach the desired terminal. Usually as one runs along the gut it suggests the popular song, "I don't know where I'm going, but I'm on my way." The stimulation of peristalsis and noting the direction of the waves I have found as helpful as the use of salt in the apprehension of birds, recommended to me in my early ornithological studies. Monks has studied this problem also. He notes that when a fold of mesentery is followed with the finger it must enter one or other fossa of the abdomen. For instance, the surgeon stands on the right of the patient holding a loop of gut parallel with the line of insertion of the mesentery, if in passing the index finger along the left surface of the mesentery, the finger enters the left fossa of the abdomen that portion of the loop nearest him will lead to the ileocecal valve. If, on the other hand, the finger, starting on the left side, describes a spiral, the surgeon knows he has the mesentery on a twist, and must reverse his ends. Monks suggests that by making traction on a loop of gut held at its extremities by the thumb and the index finger of each hand, a twist if it exists, may be determined. This is a quick way, and it is of some value when the incision in the abdominal wall is of considerable length. Following with a finger is, however, certain and is more quickly done. In very fat persons both methods may leave the operator still confused.

The Attachment of the Root of the Mesentery.—The usual line of attachment of the secondary mesenteric root is a line starting at the primary root describing a curve with its concavity upward, to the ileocecal junction. This line varies with the situation of the cecum and with the manner of termination of the distal end of the ileum. Stopnitzki has made a special study of the line of attachment. Extreme variations have been noted from a line parallel with the mesocolon directly downward to the promontory, as in a case reported by Rocher, to a line extending directly from the root of the mesentery to the ileocecal junction. The usual attachment describes a gradual curve with the concavity to the right from the root of the mesentery to the usual site of the ileocecal junction. The variation of the position of the cecum does not cause a like variation in the direction of the line of mesenteric

attachment. It is more dependent on whether or not the terminal ileum has a mesentery.

The Attachment of the Mesosigmoid.—At birth the mesocolon attaches along a line extending from the upper pole of the left kidney medially to the region of the fourth lumbar vertebra and hence downward to the sacrum. Later it becomes attached along an S-shaped line, extending from the promontory of the sacrum directly lateralward. The entire area unattached in the fetus becomes simultaneously adherent. A fetus or infant body shows either the embryonal or the adult type, never a transitional state. Samson found on the contrary, a gradual change from the fetus, the newborn, in children, and finally in the adult.

I have studied with care the process of the agglutination of the sigmoid with the parietal peritoneum. It occurs by the formation of fibrous bands between the two surfaces. The time at which this takes place is variable, but in the majority of cases it takes place between the seventh and the ninth month of fetal life. Not infrequently it is still free in newborn children. The line of attachment of the two surfaces sometimes appears scar-like. This led no less an authority than Virchow to assume that these lines were the products of inflammation. The fibrous lines noted in later life which Virchow called "peritonitis chronica mesenterialis" may bear some relation to this. As Toldt has repeatedly pointed out, there is no evidence of a coexistent inflammatory process.

Anatomy of the Ileocecal Region.—This region may arbitrarily be made to include a triangle defined by lines drawn from the umbilicus to the tubercle on the crest of the ileum and to the tuberculum pubicum. This region has for its center of interest the ileocecal junction and the appendix.

In securing their final situation the enveloping peritoneum of these structures, by virtue of their primary high agglutination with the primitive posterior wall and their subsequent sliding to their final position, suffers foldings and reduplications. The folds thus produced have been designated as folds or plicæ. Depressions included between these folds have been called fossæ, which corresponds to the fovea of the inguinal region. These fossæ are occasionally the seat of hernias. There are in addition to the regular folds many accessory ones of more or less constant occurrence. The folds

are of interest also because they have been accused in more recent years of sinister effects in limiting the function of the bowel. Much has been written dealing with this phase of the subject. That no such import can justly be ascribed to them is now generally recognized; nevertheless, it seems worth while to review the general anatomy of this region if for no other reason than to aid in preventing a repetition of this *furor anatomica* on the part of our grandchildren when they become surgeons. Some of the more accessible literature will be cited.

The folds with their resulting fossæ, of practical importance because of occasionally harboring hernias, may be first considered. The nomenclature of the folds and the fossæ is distressingly prolix. In selecting the terminology I have been guided as much as possible by usage. In adopting a name for a fold under discussion I have sought to preserve its identity by recording the name of the writer who first proposed the name. It has been found impossible to adhere to any definite scheme, however.

1. *The Ileocolic Fold (The Superior Ileocecal Fold of Waldeyer. Anterior Vascular Fold, Moynihan).*—This fold begins diffusely in the mesentery of the ileum, and descends downward to the root of the appendix or over the ascending colon (Fig. 68), and often over the cecum, particularly when the appendix is directed to the left and upward. It is usually situated over the ileocolic artery. Frequently this vessel raises up the fold to such an extent that the peritoneum forms a veritable mesentery for the vessels. When such conditions obtain a fossa is often produced,—the ileocolic fossa.

This fold has received many names. The one heading the paragraph above is selected because of usage and anatomic exactness. Moynihan, Lockwood and Rolleston, called it the ileocolic fold. The fold most commonly extends over the colon, and frequently over the cecum. The latter fact has led a number of anatomists, notably Waldeyer, to make this the prominent factor in the designation, and in order to distinguish it from another he qualified this as the superior ileocecal fold. Tuffier also adopted this name. Because of its intimate relation to the ileocolic artery it has been called the vascular fold. In order to emphasize the relation of this fold to the mesentery, Jonnesco called it the mesenteric-cecal,

and to define its topographical relation he called it the pre-ileal. This author states that this fold is always present, although it diminishes in size in advancing years. My observations have failed to confirm its constancy; in fact, not infrequently I have found only a dimple in this region. The existence of the fold is

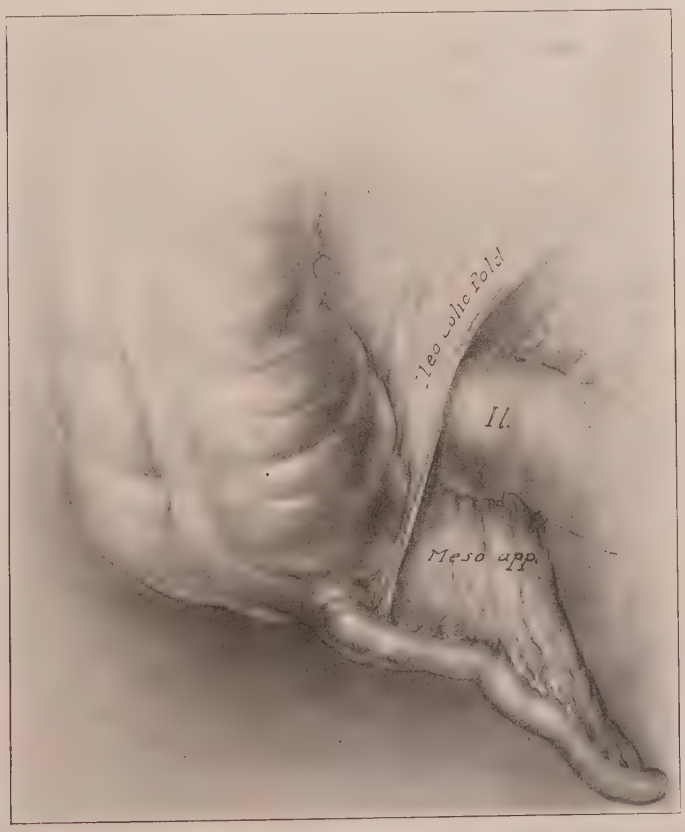

Fig. 68.—The ileocolic fold and fossa.

dependent upon the relative size of the artery in this region. If the corresponding posterior branch, the appendicular, is very large the anterior is apt to be small, and in that event the fold is small or wanting. On the contrary, if the anterior division of the vessel is large the fold is apt to be prominent. Berry and Treves likewise have found it constant.

Moynihan describes an accessory vascular fold caused by the raising of the peritoneum by the lateral branch of the ileocolic artery. When well marked it exists as a falciform fold, looking upward and laterally, forming a meson for the above-named vessel. It may form the roof of a fossa admitting the tip of the finger.

2. *The Ileocecal Fold.* (*The Bloodless Fold of Treves; the Superior Ileocecal Fold of Waldeyer.*)—This fold, first described by

Fig. 69.—The ileocecal fold. Above, the mesentery is seen to pass over the ascending colon and attach to the lateral abdominal wall.

Santorini, though it received its first accurate description by von Huschke, and its name from Luschka, extends over the terminal ileum to the cecum and appendix. Its relation to the cecum is exceedingly variable. In its simplest form it is a quadrilateral fold extending between the ileum and the appendix, the short side being attached to the cecum and the free edge forming the anterior boundary of the ileocecal fossa (Fig. 68). Sometimes this

fold is represented by a small quadrilateral plane extending between the ileum and cecum being unattached to either the appendix or its meson (Fig. 69). When this fold extends low enough to reach the base of the cecum and the root of the appendix it may carry an important, even the chief or exclusive, artery to the appendix. When such is the case this vessel may be severed and retract when the meson is cut and escape the ligature of both the meson and base of the appendix. When this fold is prominent it should receive separate attention before the appendix is removed. In its simple form it deserves the name ileoappendicular, applied to it by Jonnesco. It is usually nearly transparent and relatively avascular. This led Treves to apply the term "bloodless." Reid believes, I think incorrectly, that it extends well over to the cecum and sometimes envelops this part of the gut and extends over upon the parietal peritoneum. These broad folds when pronounced are independent of the appendix and its meson extending from the mesentery of the ileum to the parietal wall (Fig. 70). The cecum pushes into this fold carrying it before and resembles nothing more than a distant view of a plump female reclining in a hammock. In either case the occupant can with proper manipulation be extracted from the envelope. Not infrequently when the cecum has been extracted from these folds the appendix with a true fold of Treves is found. Eastman accepts Reid's view. However, one may find these broad folds enveloping the cecum covering but unattached to a true bloodless fold as described by Treves. The bloodless fold, as described by Reid, is due to the descending cecum burrowing in a parietal fold. These considerations probably induced Luschka to give this fold the name heading this paragraph. He demonstrated muscular fibers in its border, and believed that its function was to prevent the ileum from assuming a too vertical position and thus producing a kinking of the gut at the point of its entrance into the cecum.

Relative to its vascularity, Jonnesco has emphatically declared against the view expressed by Treves. Scientifically considered, he is correct, for vessels can always be readily demonstrated by injection. Nevertheless, it is strikingly devoid of vessels as compared with the neighboring folds when viewed by the naked eye. It should be noted that Treves admits that small vessels may be

noted; therefore his term must be considered relative only. Often an artery, a branch from the appendiceal, may be seen to course along its free border.

This fold in later life often receives a heavy deposit of fat which may take on finger-like projections, warranting Tuffier's suggestion that it resembles a little omentum. This comparison,

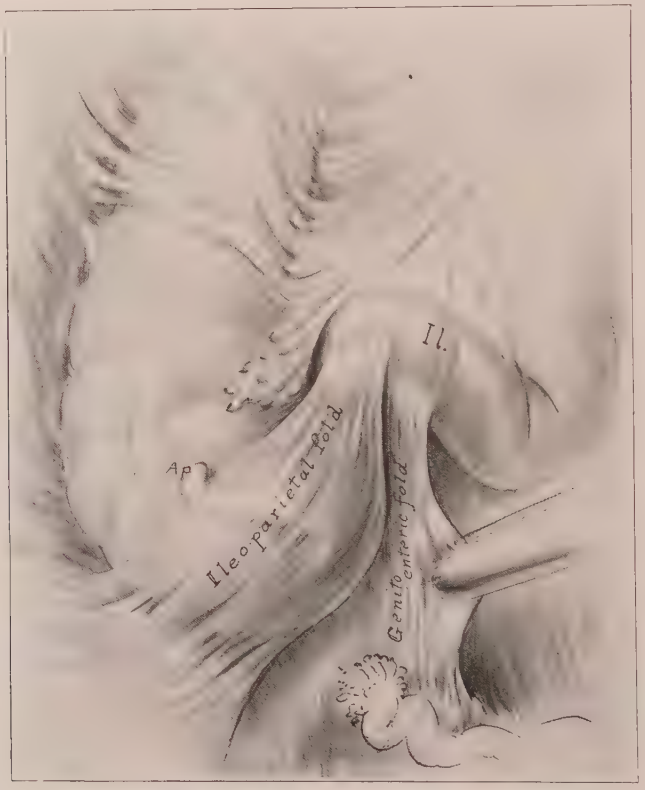

Fig. 70.—The ileoparietal and genitoenteric folds.

it seems to me, is very apt, for the fold often plays a not inconsiderable part in the walling off of incipient appendicular perforations. Lockwood and Rolleston have confirmed the presence of the muscular fibers, and have demonstrated vessels from the anterior and posterior ileocecal vessels and from the appendicular. I have frequently demonstrated branches from the anterior ileo-

colic, and recurrent branches from the appendicular; but I have never found any structures that gave the specific tinctorial reaction of muscle fibers. Clado also failed to find muscle fibers. This author proposed the term "anterior mesoappendix" for this fold.

When this fold joins the appendix the term "intermediary fold" applied by Moynihan is justified.

3. *The Mesoappendix.* (*The Posterior Vascular Fold. The Inferior Ileocecal Fold of Waldeyer*).—This is the most constant and most important fold in this region. Its importance is dependent upon the fact that it is most frequently the carrier of the appendicular artery which requires ligation in appendectomy. It is derived from the under layer of the mesentery of the ileum, and extends to the appendix (Fig. 68). Its point of origin varies considerably. Sometimes it is given off at the upper border of the intestine opposite the point where the arteria intestini attenuis divides to encircle the gut, sometimes near the opposite or free border of the gut, and sometimes it unites with the ileocecal fold, forming a sort of secondary mesentery. Not infrequently this anterior fold seems to predominate. This is probably what led Bockdalek to conclude that it may arise from the anterior layer of the iliac mesentery. He called it the "mesenteriolem."

The prominence of the appendicular artery coursing along its free border justifies the name "vascular fold." The reciprocal relation of the vessels to the mesoappendix with that of the anterior vascular (the ileocecal) fold, has already been noted. In some instances the posterior branch of the ileocolic artery is wanting, and the appendix is supplied by the anterior branch. In this event the mesoappendix is continuous with the anterior vascular (the ileocolic) fold. The chief artery, instead of being near the free margin of the meson, may lie near the base of the appendix (*1* and *2*, Fig. 71). This is likely to be true especially when the meson is rolled up itself, and the appendix points downward and lateralward. Appendices in this position may receive their blood supply from behind the cecum (*3*, Fig. 71). This is particularly confusing when there is a well marked meson between the ileum and cecum (Fig. 72). The meson may lie in front of the cecum while the blood supply comes from behind (Fig. 72). This is the most treacherous of all since the surgeon after ligating the meson

assumes that he has secured the blood supply, overlooking entirely the source from behind. In some instances an equally developed meson may lie before and behind the ileum each bearing an artery thus producing the condition found in some apes (Fig. 73).

Treves describes the mesoappendix as triangular, while Jonnesco

Fig. 71.—1. Normally situated appendix with the larger vessel situated near the base. 2. The same relation of the vessel as in the preceding, but the meson is rolled upon itself. 3. The cecum may have descended over the meson bringing the vessels retroperitoneal. 4. The entire appendix is retroperitoneal and receives its blood supply from a number of vessels.

describes it as quadrilateral. Obviously, its form depends upon the form of the cecum and the site of origin of the appendix. In those instances in which the appendix arises in the angle between the cecal pouch and the terminal portion of the ileum it is, ob-

viously, triangular. In those instances where the cecum extends downward as a symmetrical pouch and the appendix arises from its most dependent portion, obviously, it must be quadrilateral.

Not infrequently the mesoappendix is fused with the parietal peritoneum, the appendicular artery being virtually retroperitoneal (*4*, Fig. 71). When this is the case, frequently the terminal portion of the ileum is likewise devoid of peritoneum. The expla-

Fig. 72.—The vessels reach the appendix from behind and there is meson extending from the front of the cecum.

nation for this is that the fusion common for the ascending colon has extended over the terminal ileum. It is interesting to note that, when the terminal ileum is so fused, if the gut is lifted out of its normal position, a "kink" is produced.

The position of the appendix when the meson is fused is variable. Most frequently it lies in the region of the mesial parietocolic fold, but may vary either more laterally or mesially from this.

When such an appendix is lifted up, the parietal peritoneum of course follows it. If forcibly separated, the artery of the appendix may be torn, and troublesome hemorrhage result. The failure to recognize this normal position of the appendix causes operators to assume that a preëxisting inflammation has occurred. Lockwood

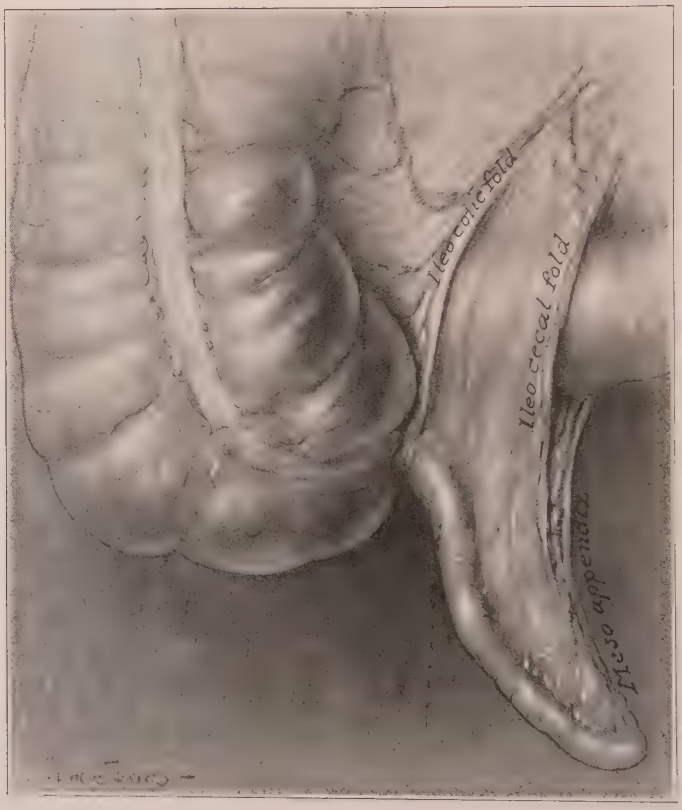

Fig. 73.—Double mesoappendix.

and Rolleston have made an elaborate classification of these more unusual locations of the appendix, but because of the great minor variations their scheme is difficult to follow.

In some cases the appendix may lie beneath the peritoneum in which instance a meson is lacking. In my observation about 1 per cent belong to this class. In some cases the appendix is retro-

cecal and retrocolic, and is unattached to the peritoneum at any point. In such cases Ferguson points out that, if perforation takes place, a retroperitoneal abscess is the result. I saw but one such in 1,087 cadavers. When the appendix is hidden it does not follow that a previous inflammation has taken place. That an appendix once intraperitoneal may become extraperitoneal from inflammation seems unlikely. Before such fusion is accepted as a fact it must be demonstrated that a parietal peritoneum exists beneath the newly formed peritoneum.

More frequently a part of the appendix is retroperitoneal while the greater portion is intraperitoneal and contains a true meson. In such cases the appendix extends upward in the region of the kidney. Inflammation of the appendix, when it is in this region, may, of course, give confusing symptoms.

As a rare occurrence may be mentioned the retroperitoneal appendices which are surrounded by the circular musculature of the colon as well. I have seen one such case (Fig. 74). In some of these cases the distal extremity may lie in contact with the liver.

The extent to which the mesoappendix extends down the organ is variable. In about 70 per cent of instances it extends down the entire length; in a lesser portion its existence as meson terminates at a variable distance from the tip. This early termination of the meson has led some observers to make the astonishing statement that, in some instances, the appendix is but partly covered by peritoneum (Fowler). While the meson as such may terminate before the tip is reached, the entire surface is unquestionably covered by peritoneum, and the appendicular artery continues as a subperitoneal plexus. In the instances in which the appendix is inclosed in a sheath of peritoneum, the appendiceal plexus above noted and a greater abundance of connective tissue indicate the line along which the meson has receded.

Not infrequently, particularly in corpulent women, the mesoappendix is the site of a literal deposit of fat, making it appear as a small omentum (Fig. 75). Often there is associated a small uniformly atrophied appendix. Microscopic examination shows nothing more than a general atrophy common in old age. Considering in association with these changes, the dense character of the surrounding fat I am disposed to believe that the atrophy of the

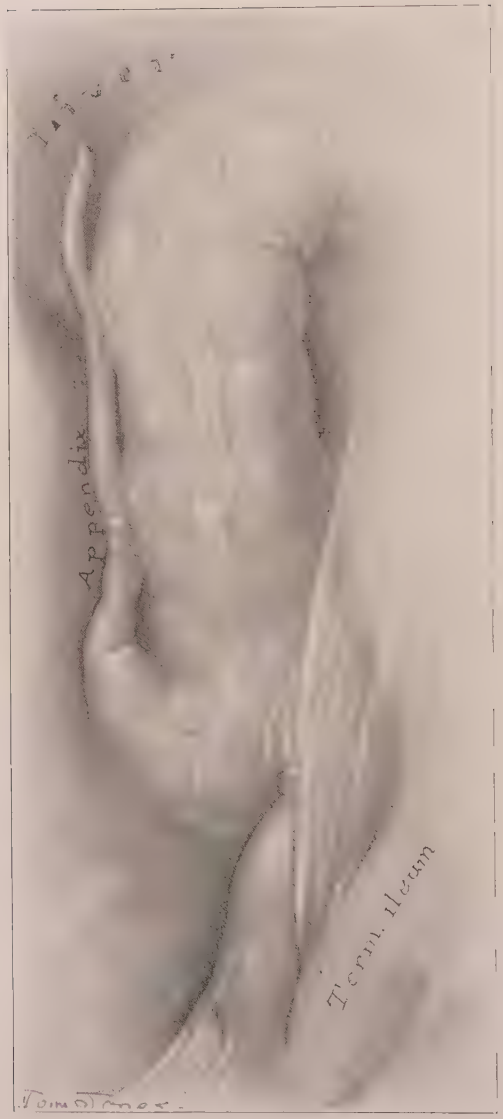

Fig. 74.—The appendix lies lateral to the ascending colon and is partly enclosed in the circular muscle fiber of the gut.

appendix is brought about by the disturbed nutrition due to the pressure from the fat.

There is no more important problem in practical surgery than the anatomy of the mesoappendix because of its association with the vascular supply of the appendix. If the meson is not sufficiently long to permit the delivery of the appendix into the wound, the incision in the abdominal wall should be lengthened sufficiently

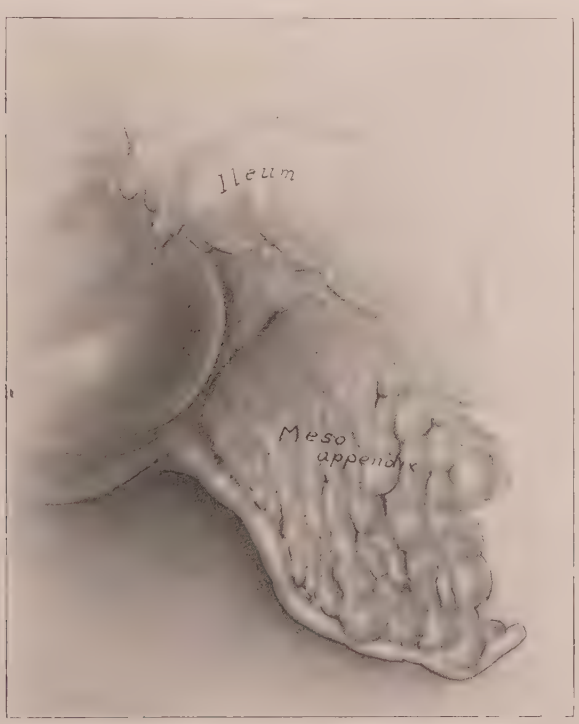

Fig. 75.—An unusually heavy deposit of fat in the mesoappendix.

to permit dealing with it *in situ*. When the delivery of the appendix causes bleeding the source of the hemorrhage should always be determined lest a continuation of it after the abdomen is closed should lead to disaster. The prime reason that surgeons take so much liberty with the mesentery of the appendix seems to be that they conceive the common type as constant. Any deviation from the type is apt to be ignored. Variations from these are develop-

mental factors and should be dealt with accordingly, and the exact source of the blood supply should be determined in each instance. The position of the appendix is dependent, let it be remembered, on its meson, and this, in turn, on its vessels. Given the position of the appendix, its meson can be predicted. This known, the location of its vascular supply can be deduced. In order that this line of reasoning shall be valid it must be understood why the appendix occupies a given position.

The importance of this knowledge is doubly vital when the organ is acutely inflamed and surrounded by temporary adhesions. The meson may be obscured, but the direction and location of the appendix declare where its blood supply must lie. In the inflamed state both it and its contained vessels are easily torn with the attendant danger. Only so much of the attendant adhesions should be loosened as will permit access to the meson. This makes accurate knowledge important.

Absence of the Appendix.—The beginner in abdominal surgery sometimes seeks refuge behind the general belief that the appendix may be absent. It is interesting to review the literature in order to determine the probability of the correctness of this view of the reason for the failure to demonstrate the organ.

Most recent writers question whether the appendix is ever congenitally absent. Dwight states that its absence has been noted by himself. Dailey collects the literature and reports a case of his own.

The only true instances of anatomic absence are those due to some anomaly of development, but there are conditions which destroy the appendix to such an extent that its demonstration is difficult. Usually when the appendix is apparently congenitally absent care will make it possible to demonstrate a rudimentary organ either as a fibrous cord extending downward from the cecum and concealed in folds of the peritoneum or upward beneath or within the muscular walls of the cecum. A microscopic section may be required to prove the existence of a rudimentary mucous membrane. When no tissues can be demonstrated in these cords the appendix may be said to be absent. In some instances a small nipple-like projection may represent the appendix. These according to Ellsworth Eliot may represent appendices epiploicæ. Hunt-

ington gives as the cause of the absence of the appendix the failure of the cecal bud to develop beyond that necessary to produce the cecal pouch. Daily suggests the contrary hypothesis, that instead of that portion normally forming the appendix failing to be arrested continues as the cecum. Schridde's case in which there were six hustra in the cecum is cited as an example. As a final cause may be cited the case of Robinson in which the cecal bud apparently failed to develop at all and the small gut was directly continuous with the colon.

The usual cause for the alleged absence of the appendix is some pathologic process. It may be so buried in old adhesions that its demonstration is difficult. More often it has been partially destroyed by necrotic processes so that a clean amputation at the cecum is produced. A cicatrix at this site then bears testimony of the past catastrophe. In one of my patients such a scar presented and a small bean-sized nodule fully 10 cm. below represented all that remained of the appendix. Even so small evidence may be lost as suggested by the case reported by Brügel. Marshall and Edwards make the startling assertion that intussusception of the appendix may be a cause of apparent absence.

About a dozen authentic cases of congenital absence have been reported. In some of these cases there was not a sufficiently thorough search made to warrant the assumption of its absence.

The Retrocolic Folds.—Extending from the cecum and the ascending colon are reduplications which attach this part of the gut more or less closely to the posterior abdominal wall. Huschke (l.c., p. 217) was the first to describe one of these under the term "ligamentum intestini cæci."

The folds about the cecum are essentially two, one extending medially and one laterally from the large gut to the posterior wall. In some cases an additional fold extends from the posterior surface of the mesentery of the ileum to the parietes. These folds are inconstant and are more subject to the personal equation of the observer than any of those heretofore considered. By grasping the appendix or most dependent portion of the cecum, and making traction, first in one direction and then in another, folds of various kinds and degree may often be made to appear. That only such folds should be noted, which are apparent when the parts

lie in their normal position, is obvious. When this conservative method is pursued the folds in this region become vastly less striking than the literature would have us believe.

The conclusions below set forth are the result of a study involving many hundreds of cadavers and an equally large number of observations at the operating table.

1. **The External Parietocolic Fold.**—This fold is formed by a reduplication of peritoneum the outer half of which is continuous with the parietal peritoneum external to the cecum and colon. The inner leaf lines the retrocecal fossa, and is continuous with the peritoneum lining the posterior surface of the cecum (Fig. 76). The distance this fold extends up the gut is variable. In many instances it is made apparent only by pulling the cecum upward and forward. This represents this fold in its simplest form, and serves to give the student a notion of its topographic relations. The constancy of the folds in this region can be appreciated only after a prolonged study. The unimportance of these variations can best be appreciated by studying the descent of the cecum in the fetus.

In order fully to understand the relation of the peritoneum to these folds, it is necessary to study the subperitoneal connective tissue apart from the peritoneum covering it. If the peritoneum is carefully removed there remain in well-marked instances fascial bands, which serve to anchor the cecum in its place, warranting the term "ligamentum colicum dextrum" applied by Hensing and "ligamentum intestini cæci" by Huschke, and "superior ligament of the cecum" by Tuffier. The fascial bundles extend from the ascending colon and the cecum to the fascia of the iliac fossa, over the false capsule of the kidney even as high as the attachment of the diaphragm, warranting the term "ligamentum pleurocolicum" applied by Langer. Those instances in which the fibers are particularly prominent below are the only ones in which a distinct fold appears below. This fold bears a certain resemblance to the broad ligament of the uterus so far as its suspensory capacity is concerned, and, when prominent, its free border is not unlike the ligamentum infundibulopelvicum. In that event it may form the lateral wall of a complete pocket behind the cecum.

Sometimes this fold forms a sort of pocket for the cecum.

Huschke evidently had this type in mind when he described it as follows: "It arises sickleform from the fascia of the ileac muscle of the right side forming a sacculated groove which takes in the cecum." These folds are relatively avascular, and have been referred to by Reid apparently without warrant as "bloodless folds of Treves."

Independent bands may exist extending from the cecum or the colon to the lateral abdominal wall. These may exist as real lig-

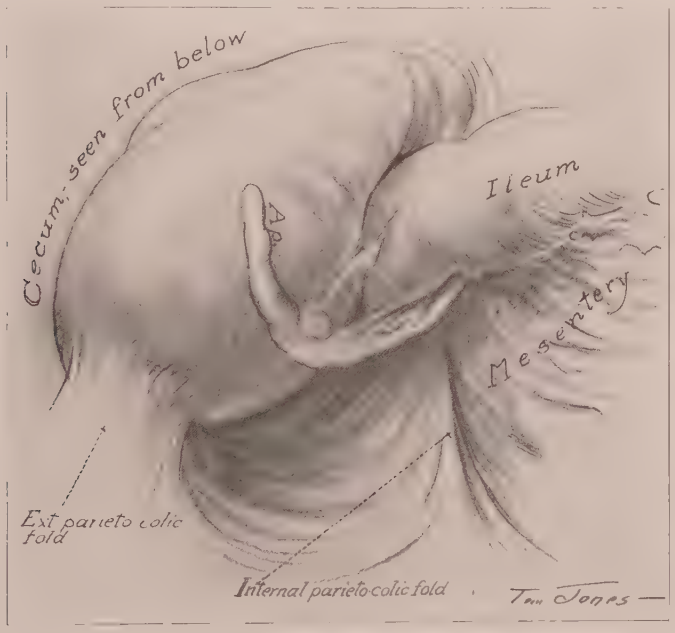

Fig. 76.—External and internal parietocolic folds.

aments, permitting a space between them and the posterior wall (Fig. 77), or, as is more commonly the case, they are fascial bands carrying the peritoneum on their summit. These may extend directly lateralward at right angles from the abdominal wall, or they may extend mesially and downward depending on whether or not the attachment was formed before or after the cecum has reached its final abode. These folds may assume the form of an adventitious band. This is particularly likely to be the case when from some irritative process the potential vessels become dilated.

Pathologic significance was at one time ascribed to these by Jackson (see Pathology of the Circulation).

In rare instances the great omentum may become attached to the ascending colon, even passing over it and becoming attached to the parietal peritoneum, as was first described by von Haller. This condition has been regarded as pathologic, and Eastman describes a case in which acute obstruction was associated with it. Sometimes the mesentery receives an additional deposit of subperitoneal fat, which may resemble the edge of an adherent omen-

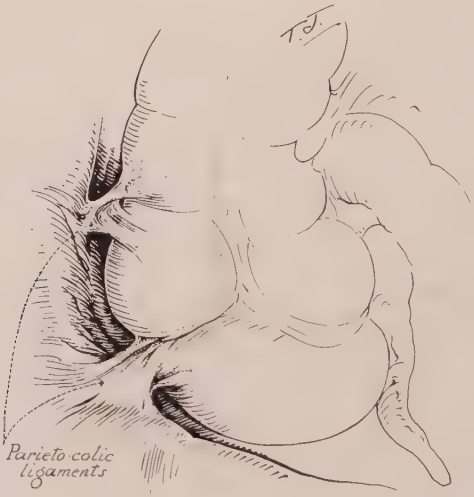

Fig. 77.—Parietocolic ligaments.

tum. This is most likely to take place in the ileocecal juncture (Fig. 78).

The deeper fibers of the superior colic ligament whether a fold of peritoneum has been defined or not, have been gathered together as the nephrocolic ligament by Longyear. These, no doubt, do have the function of holding up the cecum, though it is somewhat a stretch of the imagination to call them ligaments.

Most of these folds are more or less subjective since they appear only when traction is made on a particular segment of the gut. That they even are factors in occluding the lumen of the gut has not been demonstrated. The only way to get a comprehen-

sive picture of this region is to examine a large number of bodies in which the viscera have been fixed *in situ*.

2. **Internal Parietocolic Fold.**—This fold bears the same relation to the medial border of the cecal pouch that the foregoing does to the lateral border (Fig. 76). It is much less constantly present because of the variability of the attachment of the terminal ileum to the posterior parietal layer. When it is perfectly formed, this, in association with the external, forms the retrocecal fossa. This one more frequently than the external requires traction on the

Fig. 78.—Heavy deposit of fat in the mesentery of the ileocecal junction causing it to resemble an adherent omentum.

cecum before it comes into evidence. This fold is seldom well marked except in children.

3. **Mesentericoparietal Fold** (Jonnesco).—In contradistinction to the external parietocolic fold, Tuffier called the mesenteric parietal the inferior ligament of the cecum. Engel seems to have been the first to describe this fold under the term "plica intraangularis." When the parietocolic fold is absent or illy developed or very rarely, when it is fully formed, a fold may exist which extends from the posterior leaf of the ileac mesentery to the posterior parietal peritoneum. This is particularly prominent in the fetus. It is then

often a prominent fold which looks like a gubernaculum. When this fold remains permanently it represents the genitomesenteric fold of Reid, and when the unfolding is especially complete at its medial termination it forms the genitomesenteric band (the plica genitoenterica) in the Treitz sense. In such cases the terminal ileum may be in part retroperitoneal.

Reid in studying the fetus, found a fold extending from the region of the duodenum to the genital gland, in eleven of twenty fetuses examined. These fetuses were from 12 to 20 cm. cephalococcygeal length. He describes the fold as passing downward from the inferior surface of the mesentery in the right half of the abdomen, dividing the abdomen into two compartments. The right compartment is the smaller and contains the cecum, while the left is larger and contains the pelvic colon. The boundaries are defined by it posteriorly to the abdominal wall along the line of the spermatic or ovarian vessels, while the anterior border is continuous with the inferior surface of the mesentery, generally along the line of the ileocolic artery. This anterior border is free, and is continuous with the suspensory ligament of the ovary.

This fold in my experience is present in the adult only if there has been a failure, in whole or in part, of the cecum to become attached or to descend. The sole interest in this band lies in the light it throws on the method of descent of the cecum and in the formation of the plica genitoenterica of Treitz. This fold occurs when a part of the terminal ileum is retroperitoneal. In such instances, by lifting up on that portion of the gut which has been deprived of its mesentery, a fold extending into the pelvis may be demonstrated.

For a time a clinical significance was ascribed to the structures. If the cecum is pulled violently upward the ileum may be made to show a kink at the point of attachment of this fold, to which Lane and his followers have attributed a pathologic significance. It is worthy of note that a kink is produced only when the structures are pulled out of their normal position. At this time it seems undesirable to review the astonishing array of literature that appeared, for it had as a purpose only to form a "scientific" basis for a none too commendable clinical speculation.

At the time the fold of Reid appears in the fetus it is to be

remembered that the development of the peritoneum is not complete. On the contrary, the fold is formed of embryonal connective tissue covered with a layer of undifferentiated mesoblastic cells. When fusion of the peritoneum of the cecum and transverse colon with the parietal peritoneum occurs, it is not as a developed peritoneum, as the schematic drawings of the books would seem to indicate, but as a gelatinous mass as yet but slightly differentiated into cells, membranes, and connective tissue, as already pointed out. The same is true of the schematic drawings of Reid. Instead of being a sheet, it is a pyramidal mass. As the descent of the cecum occurs this mass flattens out as the cecum descends somewhat as the walls of a tent descend when the central pole is removed. It is the degree of this spreading out of the fold that determines the end-relation of the cecum to the posterior abdominal wall. If this does not occur at all a mobile cecum and ascending colon result, and the colon retains a complete mesentery. When nearly complete the lateral parietocolic ligament is not marked and the medial colic ligament is absent or imperfectly developed while the mesenteric parietal fold of Jonnesco is present. When fully developed this fold is absent while both parietocolic ligaments are present, and the ileum retains a complete mesentery to the point where the appendicular artery reaches the meson.

What was said in the chapter on development regarding the attachment of the cecum to the posterior abdominal wall may be repeated here. The old theory that the colon becomes retroperitoneal still remains true in part insofar as the cecum is concerned. The cecum usually becomes attached at or near the upper pole of the kidney, and here becomes retroperitoneal. At this early stage there are no colic ligaments nor any retrocecal fossæ, but the peritoneum falls over the terminal portion of the cecum like a blanket over a pumpkin. As development progresses this blanket-like covering, together with the cecum, descends. As the gut exceeds the peritoneum in its rate of development the cecum pouches and the ligaments in question become prominent, and a fossa between them may be produced. This blanket-like peritoneum has really not yet taken on the structure of peritoneum, but is made up of a colloidal-like mass of cells. In the process of attachment the budding appendix may be engulfed, and a retroperitoneal appen-

dix result, which in some instances takes its nutriment from secondary vessels, and the mesoappendix as such does not develop. At the time the colon makes its migration it is very small relatively, being less in diameter than the jejunum. As it increases in diameter it makes a requisition on the surrounding peritoneum, which, if it is unequally attached to the parietes, responds in varying degree in the different parts of the gut.

In some cases it is true that the cecum behaves as the schematic drawings of the books would have it do, namely, remains unattached until it reaches the iliac fossa; but this is rarely the case. In rarer instances the cecum fails to attach at all, and the ascending colon retains a complete meson, as in the normal state in many of the lower animals. A correct view of the nature of the attachment and of the migration of the cecum can be obtained only from the study of a large number of fetuses.

Cecal Fossæ.—With the preceding discussion of the various folds in this region, a description of the fossæ they bound may be brief. As already noted in the discussion of cecal folds, the nomenclature of the structure in this region has suffered a needless complication because of a prolix and often contradictory nomenclature. Being situated between these folds, the fossæ naturally suffer a similar fate. In adopting a nomenclature the same plan as set forth in the discussion of the folds has been adopted.

1. **Ileocolic Fossa.**—This fossa was first described by Luschka. It was called the superior ileocecal fossa by Waldeyer, a name regarded as most suitable by Treves, Hartmann and Tuffier; the recessus ileocecalis by Broesike and the anterior ileocolic or parietal by Jonnesco. The name herein adopted was suggested by Lockwood and Rolleston, and is preferable because it emphasized its predominant location at the ileocolic fold in front and the ileum and its mesentery behind. At its base is the cecum. Its opening points mesially and downward. When the mesentery of the appendix is derived from the ileocolic fold, this fossa extends well over the terminal portion of the ileum, and then may be large enough to admit the terminal phalanges of two fingers. This fossa is usually well marked in children, is likely to be much reduced in advanced years, and is often obliterated, particularly in obese subjects.

Moynihan describes an accessory fossa, the accessory anterior vascular fossa found above the ileocecal fold. It occupies the angle between the colon and its mesentery, being roofed over by the accessory membrane. It is only occasionally present.

2. **Ileocecal Fossa.**—This fossa was first described by Huschke, but the term ileocecal was suggested by Luschka and adopted by Lockwood and Rolleston, Kelynack and Berry and others. Waldeyer added the prefix interior to distinguish it from the ileocolic which he called the superior ileocolic. This lead was followed by Laenet, Treves, Tuffier. Clado called it the very appropriate name of ileoappendicular fossa which Moynihan adopts. In cases in which the ileocecal fold becomes united with the mesoappendix (see p. 84, Fig. 29, Moynihan) this term is the only correct one. It lies below the ileocecal fold (bloodless fold of Treves) and above the mesoappendix. Above is the ileum; and its lateral border, the apex of the fossa, is formed by the head of the cecum. This fossa is often well marked in young persons; but it is usually obliterated by the deposition of fat in persons of advanced years. It is often well marked even in adults, frequently being large enough to harbor a hickory nut. Two cases of cysts (reported by Schott) found in this region have been explained on the supposition that the mouth of this fossa had been obliterated, resulting in the collection of fluid.

3. **The Retrocecal Fossa.**—(of Jonnesco; fossa cæcalis, Huschke and Waldeyer). This was called the retrocolic fossa by Moynihan. The term here adopted was suggested by Jonnesco, and in slight modification, "subcolic" by Lockwood and Rolleston. When it extends high up it justifies the term "retrocolic," adopted by Treves and Berry, and in its Latinized form "recessus retrocolic" by Broesike. It lies behind the cecum, by which it is bounded in front; on either side are the parietocolic folds, and behind is the parietal peritoneum. This fossa is often of considerable depth, sometimes admitting the entire finger. The depth, however, is in a measure dependent upon the degree of traction exerted upon the cecum in demonstrating it. This fossa is sometimes divided into two by a secondary fold (Jonnesco).

4. **Fossa Retroappendicularis.**—This fossa, first described by Hartmann, received the name here adopted from Leichtenstern. Hart-

mann, who first described this fossa, called it the fossa ileocecalis infirma, while Broesike called it the fossa of Hartmann after its discoverer. When well developed the fossa may extend well behind the terminal portion of the ileum, and then deserves the term recessus postilica, applied by Tarenetzky. This fossa is inconstant, but is often represented by a depression between the plica infra-angularis of Broesike and the mesoappendix. Moynihan speaks of it as an exaggerated dimple. This fossa is one of interest because it was once the site of a hernia (reported by Snow).

The Genesis of the Ileocecal Folds and Fossa.—Waldeyer was the first to emphasize the importance of the vessels in the development of the folds under discussion.

Before the budding of the cecum, the meson carries vessels to the region of the future cecum. When the cecum buds, the vessels are stretched across the added area from their origin in the common mesentery. As the cecum elongates there is an angle between the terminal ileum and the nearly developed cecum. The vessels strike the shortest line between their origin and their termination. In doing so they drag the peritoneum with them, forming a pocket between these two folds and the angle formed by the ileum and the budding cecum. As the appendix develops it secures its nourishment from one of these vessels, usually the posterior, and continues to extend below the cecum. The result is that the fold carrying the appendicular blood supply becomes the greatest and forms a meson to the organ; the mesoappendix, therefore, becomes synonymous with the (posterior) vascular fold. The extent of development of the anterior fold depends upon the degree of elongation of the cecum below the point where the vessel in question finds its primitive lodgment.

As the cecum extends below its point of beginning at the primitive gut, it carries with it bands of fibrous tissue in an early state of development. These bands carry the peritoneum before them, producing the ileocecal fold. Whether this fold is attached to the mesoappendix or to the cecum depends upon the point of attachment when the process of attenuation begins. That this fold is formed after the mesoappendix is formed is proved by the fact that when it possesses an artery it is derived from the appendiceal.

An appendix occasionally is observed which has retained two

mesons, one from the posterior and one from the anterior. This is the case when the appendix remains short. Diminutive appendices, those an inch long or less, in my experience usually are those doubly provided with a blood supply.

Huntington has called attention to the fact that in Ateles ater (the black-handed spider monkey) the anterior and posterior vascular folds remain almost equally developed. The appendices in these animals closely resemble the diminutive appendices in the human above referred to.

Treves believes that the intermediary fold is the true meson, because its peritoneum is continuous with that over the gut and its mesentery. On either side of it, however, are narrower vascular mesons. The relation is not different from that in the human, except that the relative size of the various structures varies according to the development of the cecum. A very striking "intermediary fold" may be formed in end-to-side intestinal anastomosis.

Moynihan regards the ileoappendicular fold as of twin origin, primarily from the ileoappendicular muscle and from the ileoappendicular artery. That the artery does not have a genetic importance is evidenced by its inconstancy. The secondary vascularization of artificially produced "intermediary folds" throws additional doubt into Moynihan's theory.

Why but one-half takes up the task of supplying blood to the appendix may be explained by the fact that the appendix does not go out equidistant from the anterior and the posterior lines of the cecum. This may be surmised from the appearance in the adult, and may be proved by section of the cecal buds. It may be stated as a general postulate that the nearer the appendiceal buds to the apex of the cecum the more equal are the anterior and the posterior folds.

Why the appendix buds from the posterior rather than from the anterior aspect of the cecum finds an interesting suggestion in comparative anatomy. In Cercopithecus sabræus (African green monkey) the dorsal aspect of the ascending colon is adherent to the posterior parietal peritoneum. Huntington remarks that by this adhesion the posterior ileocolic branches find an element favorable to their development. Studies in early embryos confirm this interesting surmise. When the cecum attaches early in the

subhepatic or upper renal region, the appendix is farthest posterior, and, in consequence, the posterior vascular fold is likely to be the most prominent. It is only when the cecum descends a considerable distance before becoming adherent to the posterior parietal peritoneum that the anterior vascular fold has an equal chance for development with the posterior. I have seen instances in which the appendix possessed two mesons equally developed. It possessed then both the superior and the inferior vascular folds, representing the state normal to the spider monkey.

The studies of the genesis of the cecal folds and fossæ have their practical importance, in that they show the greatest variation the various folds may assume in the normal development of this portion of the gut. When the surgeon meets with a variation it should excite, first of all, his biological curiosity, and not at all his surgical activity.

The Retroduodenal Folds and Fossæ.—The folds and fossæ in this region, unlike those of the ileocecal region, are of importance more on account of the lesions associated with them than for the understanding of the anatomy their study conveys. Hernias occurring in this region are not common maladies, but when encountered they are so perplexing that unless the surgeon has some previous knowledge of their peculiarities his manipulations are apt to result disastrously for the patient. For this reason a somewhat detailed account of them seems justified.

Hensing was the first to mention the folds about the duodenojejunal flexure and the fossa they surround. The first real description was supplied by Huschke. His descriptions as far as they go may be accepted at the present date.

The percentage of bodies in which the various fossæ are found in this region is chiefly a matter of personal equation, dependent upon the method of search. If the intestines are lifted out of their bed the existence or depth of fossæ is often proportional to the degree of traction made upon the gut about which the fossæ lie. Virchow (personal communication) is entirely correct in his contention that, in order to study these fossæ correctly, all the viscera should be allowed to lie in their normal position. If fossæ are found under these conditions they may properly be recorded as being present.

In the following discussion Broesike's classification will be followed as nearly as possible. This is done because he has presented the subject in a clear manner in his book and because I obtained a bias in his favor by often listening to his clear demonstrations. His presentation has received at my own hands some modification based on anatomic studies in the autopsy room and particularly from studies in the living subject. Instead of the ponderous Latinized terminology employed by Broesike, I have followed the Anglicized equivalent as nearly as possible.

The Duodenal Folds.—The folds in the region of the duodenojejunal flexure are due to the fact that the duodenum, a retroperitoneal organ, becomes at this point an intraperitoneal one, the jejunum. In so doing folds result in the peritoneum at this point. Broadly speaking, these folds are developed one on either side of the duodenal flexure, one below and one above. The exact relation these folds bear to the segments of intestine in question depends in large measure on the position of the gut, as already mentioned. The best recognized of these folds are the superior and the inferior duodenal folds. When both of these are present an ellipse or circle is formed (Fig. 79). The older authors gave very accurate descriptions of these folds. Thus Monro speaks of them as forming a ring and Haller and Sandifort speak of them as forming an arc of a circle. Huschke speaks of these folds as two sickle-like folds which extend from above and below where they unite.

The relation of these folds to blood vessels in this region is a matter of great importance, to which Treitz first called attention. According to him the upper limb of the fold is defined by the inferior mesenteric artery the two forming an arch. These vessels furnish the keynote to the correct understanding of the fossæ in this region.

As a rule the "arch of Treitz" can be demonstrated. Near the upper horn of the semilunar fold the interior mesenteric vein can be found by dissecting off the peritoneum if it is not apparent at a glance. Below and usually at some distance from the free edge of the fold the inferior mesenteric artery is found. The artery instead of coursing upward to cross the vein often arches downward and outward. In that event a true ring is not formed.

Numerous theories as to the origin of this fold have been ad-

vanced. Treitz regarded them as traction folds. Moynihan objects to this theory because of the early attachment of the duodenum by the suspensory muscle, which negates any traction. It must be remembered, however, that a traction fold may be produced by the movement of but one end of the line. The movement of the colon can hardly be accused, for the folds may be present before the final fixation of the splenic angle. However, the meson at its mesial termination becomes fixed at an earlier period, and from this point traction may be made. When first formed the "ring"

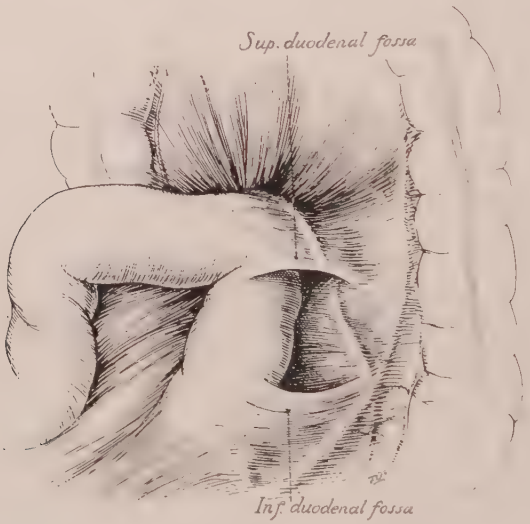

Fig. 79.—Superior and inferior duodenal folds and fossæ. (Copied from Poirer and Charpy.)

about the flexure is small. As the duodenojejunal angle extends upward in the process of growth, and the point of attachment of the lower fold extends downward as the splenic fold of the colon to which it is attached increases in length, the two points must of necessity become separated. This factor, acting in conjunction with the retarding action of mesenteric vessels, according to Waldeyer's theory, it seems to me, furnishes the most plausible explanation. Waldeyer's theory is that the folds form a mesentery for the inferior mesenteric vein. While, as a matter of fact, in many of the best developed examples of this fold, the vein in

question is not found at the free border but this does not prove that it did not occupy this position in the embryo. Therefore, the contention of Eppinger that a vein does not always bear a close relation to the fold, does not negate the theory. The shifting of the mesenteric vein, no doubt, is possible, not only after the fifth month, at which time these folds most frequently appear, but in subsequent life, when the statics of the abdomen change from the increase or lessening of the mesenteric fat.

Infrequently the fold lies far to the left of the duodenojejunal angle, and then forms a distinct meson for the inferior mesenteric vein. Even Moynihan, otherwise an opponent of this theory, accepts this as a mesentery for the inferior mesenteric vein.

Treves was of the opinion that these folds represent the remains of the mesoduodenum being brought about by the unfolding of that structure. This unfolding takes place, but is an identification of the specific factor referred to by Treitz as traction. Moynihan regards them as fusion between the anterior layer of the mesentery of the ascending portion of the duodenum and the anterior surface of the meson of the descending colon.

Much less frequently there is a fold extending to the right of the ascending duodenum. This fold has the superior mesenteric artery on its free border and is, therefore, a true vascular fold.

In some instances there are two folds extending from the jejunum downward on either side of the duodenum, not unlike similar folds about the cecum. These are united below, defining a U-shaped crest (Fig. 80). This fold is accentuated by pulling downward upon the root of the mesentery or by lifting up on the jejunum. A disparity of growth of these two structures, with a greater rapidity of growth on the part of the mesentery, would seem to explain the folds in this region, just as an extension of the peritoneum over the iliac fossa after a fixation of the cecum explains the formation about that region. Two folds of peritoneum may sometimes be seen extending upward from the duodenal loop. These are the duodenomesocolic ligaments.

There is a fold extending to the right of the duodenum along the superior mesenteric artery but it is rarely observed.

The description of the fossa which is to follow is an attempt to present the essential facts in a comprehensive manner. The lit-

erature on this subject is amazingly complicated, and the student who hopes to confirm the statements contained in it must needs have great industry, an abundance of material, and a facile imagination.

1. **The Superior Duodenal Fossa.**—There is some question of the advisability of discussing separately the several portions of the duodenojejunal fossa of Treitz. The chief justification for a separate consideration lies in the fact that the two portions are sometimes unequally developed, or one portion may be entirely absent.

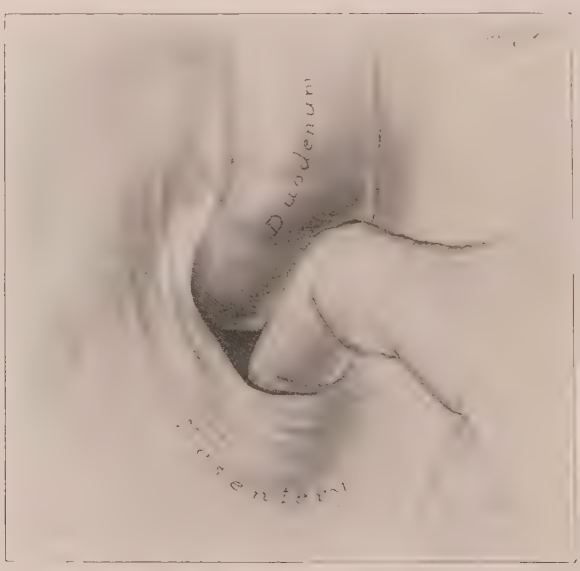

Fig. 80.—The posterior duodenal fossa is made more prominent by downward traction with the finger.

The fossa under discussion here represents the upper portion of the duodenojejunal fossa (Fig. 79). It is bounded above by the superior duodenal fold, which extends from the duodenum and is lost in the descending mesocolon. The ascending limb of the duodenum lies medially, and behind the parietal peritoneum at the level of the third lumbar vertebra. Laterally it bears a close relation to the inferior mesenteric vein. This fossa is often deep enough to admit the first joint of the finger, as I have repeatedly

observed when searching for the loop preparatory to doing a gastroenterostomy.

2. **The Inferior Duodenal Fossa.**—This represents the fossa originally described by Treitz. This is the most frequent of the fossæ in this region, and is noted in some 10 per cent of the bodies examined. However, by making traction on the jejunum upward and to the right, it may be demonstrated in 60 to 70 per cent of the cases.

In typical cases a sharp semilunar fold, the intraduodenal be-

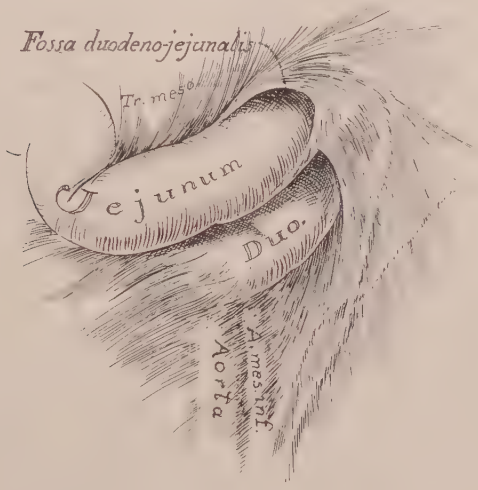

Fig. 81.—The posterior duodenal fossa. (Modified from Moynihan.)

gins below the flexure, extending from the ascending limb of the duodenum to the left, and is lost in the mesentery and posterior parietal wall at the point where the superior mesenteric vein passes under the pancreas to reach the portal vein. The fossa extends directly downward, or inclines a little to the right. In well-marked cases it may hold the terminal phalanx of the thumb. The left colic artery is usually near this fold, but downward and to the left. Microscopic section of the fetus from the fifth to the eighth month shows more emphatically the relation of this vessel to the fold in question than does the gross anatomy of the adult.

When both the superior and the inferior duodenal fossæ are

present their outer limb may be joined. When this is done a fossa of considerable extent is formed. It would seem that this type would invite more the formation of hernia than any of those to be hereafter mentioned. A study of the anatomy of the recorded cases of hernia in this region lends strength to this view.

3. **The Posterior Duodenal Fossa.**—(Recessus duodenojejunalis posterior of Broesike, the duodenojejunal fossa of Jonnesco.) (Fig. 81.) This fossa was first described by Gruber. It lies behind the upper portion of the ascending limb of the duodenum, and the opening to it lies just above the duodenojejunal flexure. It has on either side of it two folds of peritoneum, the plicæ duodenales superior (Broesike), which extends over the duodeno-

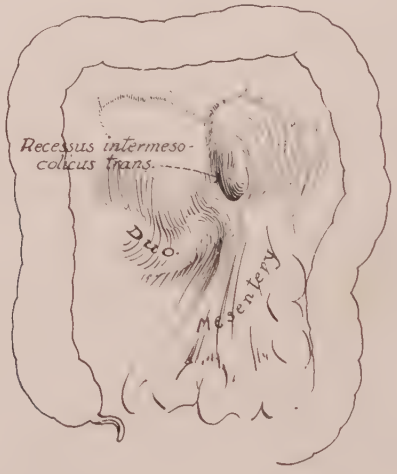

Fig. 82.—The intermesocolic fossa. (Redrawn from Brösike.)

jejunal flexure as the pillars of the fauces extend over the tonsils, and forms a small pocket behind the terminal portion of the duodenum. This same point sometimes presents an opening, which extends upward. This usually is described separately as the duodenojejunal fossa of Huschke. It is bounded on the right side by the aorta and the suspensory ligament of Treitz (or this ligament may divide it into two compartments) and to the left is the kidney. This fossa extends upward, usually somewhat to the left, and may reach as high as the pancreas. A dimple in the region is of fre-

quent occurrence, but a distinct fossa large enough to admit the end of the finger is rare. It is easy to search for this fossa when picking up the loop preparatory to a gastrojejunostomy. Moynihan believes that this fossa is most complete when associated with the paraduodenal fossa. He explains that its formation is due to the fusion of the duodenojejunal flexure and the adjacent portion of the duodenum with the under layer of the transverse mesocolon.

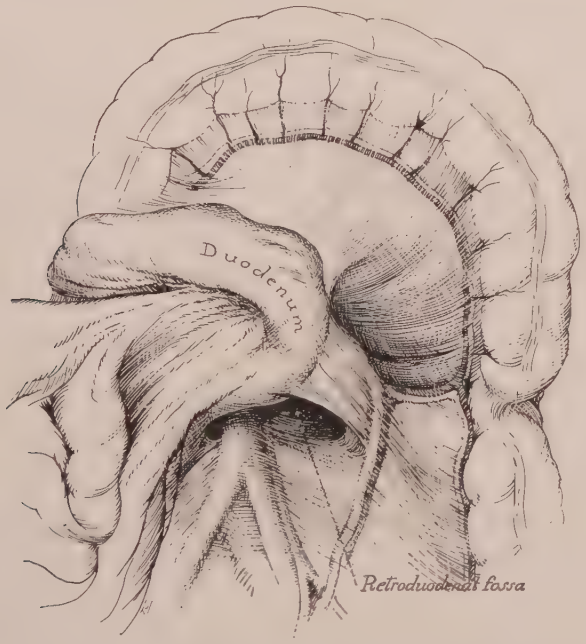

Fig. 83.—The retroduodenal fossa. (Redrawn from Landzert.)

4. The Intermesocolic Fossa.—(Broesike.) (Fig. 82.) Broesike believes this fossa is a modification of the fossa just described. He has seen it three times on the right side and an equal number of times on the left. The fossa extends either to the right or the left from the duodenum into the mesocolon, being covered by the plica intramesentericolica transversa, as Broesike calls a fold extending from the transverse mesocolon and the line of attachment of the mesojejunum.

5. **The Retroduodenal Fossa.**—(Jonnesco.) (Fig. 83.) This fossa

is bounded in front by the ascending portion of the duodenum, laterally by two parietoduodenal folds, and posteriorly by the parietal peritoneum, behind which lies the aorta. It is often very deep, extending upward to the pancreas. I regard it as an unusually well developed superior duodenojejunal fossa, in which the lower border extends downward and the ascending portion of the duodenum is unusually mobile.

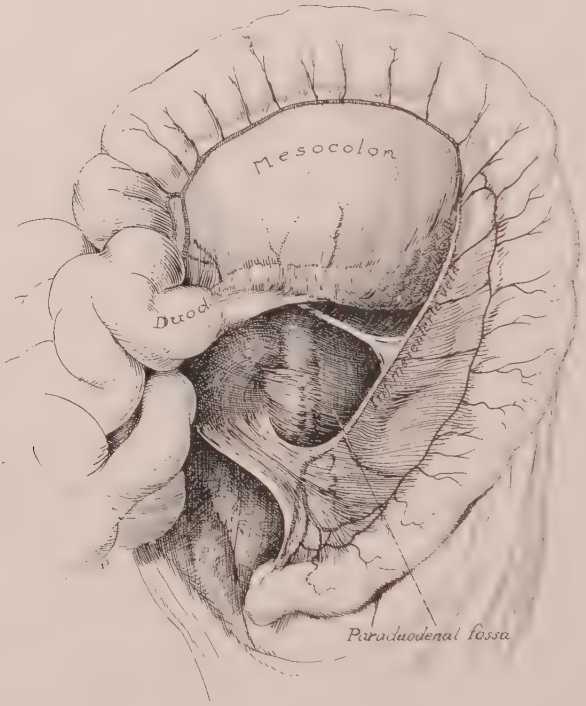

Fig. 84.—The paraduodenal fossa. (Redrawn from Landzert.)

6. **The Paraduodenal Fossa.**—(Landzert.) (Fig. 84.) This is the most artificial of the fossæ. It corresponds fairly well with the left colon recessus of Waldeyer. It is a pocket formed by the fold of peritoneum covering the inferior mesenteric vein, the plica venosa. It is most strongly marked when the colon is pulled strongly upward. It is seen in fetuses in the course of colonic rotation. I believe it is this fold growing over the coils of in-

testines early in fetal life that produces the hernias (vida infra). I have never seen it in an adult unless artificially produced by traction. It seems to be but a modification of the supraduodenal fold when the vein lies far lateralward.

7. **The Mesentericoparietal Fossa.**—(Waldeyer.) (Fig. 85.) This fossa, according to Waldeyer, is caused by the raising up of the peritoneal fold by the superior mesenteric artery as it courses downward and to the right. It may be compared in its topography with the paraduodenal fossa. Like the preceding I have found

Fig. 85.—The mesentericoparietal fossa (the right paraduodenal fossa). Redrawn from Haasler.)

this fossa only in the fetus or newborn. Moynihan has not met with it in adults.

8. **Parajejunal Fossa.**—(Broesike.) This is formed, according to Broesike when the jejunum is adherent for a longer distance than usual to the posterior wall. It is bounded on either side by folds of peritoneum "the plica parajejunalis" (Broesike) above by the jejunum and behind by the parietal peritoneum. The cavity points downward and to the right. It is emphasized by lifting the jejunum sharply upward and at the same time pulling the mesentery downward. It is rarely found, according to my experience, when the gut lies in the normal position. I have seen but few well-marked examples.

The question of the relative importance of these various fossæ in relation to hernia of this region has been much discussed. Treitz naturally enough, thought that the fossa described by himself was the most important recipient of hernias, and most writers since have not seen fit to doubt this view. This is a matter of no great interest. Moynihan reaches the kernel of the problem when he lays down the general law that the orifice of a hernial sac in this region is always vascular. According to him the inferior mesenteric vein is always found in the orifice of the sac, and, therefore, the paraduodenal fossa is the one which harbors the hernias of this region. The ultimate anatomic findings are, it may be granted, as Moynihan indicates. According to this view the hernia may begin in any one of the fossæ described above, and as the intestinal contents of the hernia increases the orifice dilates until the more rigid mesentery of the vein is reached, where further stretching of the orifice stops. It would be impossible in such instances, where the end stage only is observed, to determine the point of origin. From a practical point of view, Moynihan's observation is all that concerns the surgeon when confronted by hernia in this region. The important point to remember is that the constricting ring is very apt to harbor a vessel of importance, the severance of which would deprive a considerable area of gut of its nutrition.

9. **Intersigmoid Fossa.**—(Fig. 86.) This fossa was first described by de Haen and later by Treitz and Eve. At the inner margin of the psoas magnus muscle where it is crossed by the mesosigmoid, a dimple is frequently seen, often a veritable pouch, and occasionally a considerable sac. I have studied the genesis of this fossa in many fetal cadavers, and have observed that in them a considerable dimple is common. This fossa is evidently the result of an incomplete agglutination of the mesosigmoid with the parietal peritoneum. The point where this agglutination is most apt to be defective is the region of the common iliac artery, but may occur at any point. Several openings may occur. Broesike observed an instance in a male of thirty where there were four openings. Gruber has seen two separate fossæ and one in which there was one opening and two branches. These openings may extend upward for a varying distance. Broesike describes one extending upward as far as the pancreas.

The Foramen of Winslow.—The opening between the greater and lesser peritoneal cavities is in a way analogous to the fossa now under discussion. Unlike these, it is constant unless obliterated by inflammatory processes.

This opening is bounded above by the caudate lobe of the liver, in front by the lesser omentum containing the bile-duct, portal vein, and hepatic artery and below by the duodenum and hepatic

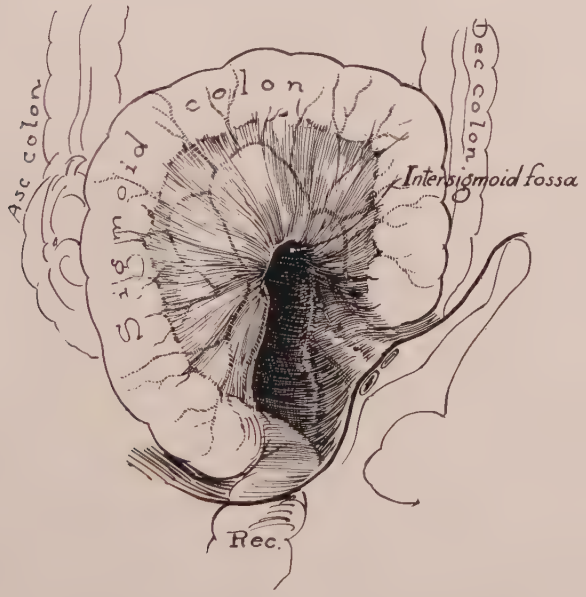

Fig. 86.—The intersigmoid fossa. (Redrawn from Moynihan.)

vessels and by the vena cava. It usually admits the tips of one or two fingers.

Hernias into the Retroperitoneal Spaces.—Hernias into these spaces are, on the whole, rare. Jonnesco collected the literature up to the time of the publication of his book.

Hernias of the Ileocecal Region.—About a dozen cases have been published. In none of these is the description very convincing. In Tuffier's case, for instance, an eight-inch coil of intestine lay in an open pouch behind the cecum. I observed such a case in the dissecting room, but it did not impress me as being worthy of

classification as a hernia. Little's case evidently was a strangulation due to the slipping of a gut through a hole in the mesentery. In most of the cases data recorded are too meager to permit an understanding of the exact anatomic relations.

Duodenal Hernias.—In contrast with the preceding a number of very clear cases of hernia have been described. Some sixty-odd cases in all have been described. They may be divided into those

Fig. 87.—Duodenal hernia. (Redrawn from Treitz.)

occurring on the right, and those on the left, side of the duodenum. In size they vary from a small nodule of gut to the entire small intestine. Those occurring on the left side present loops to the left and above the duodenojejunal angle with the mouth looking to the right. When large the descending colon may be pushed lateralward or is carried on the summit of the hernial mass (Fig. 87). On the right, likewise, the colon may be pushed

lateralward or is carried on the summit (Fig. 88). Those occurring on the left side are the more numerous. Moynihan records fifty-seven on the left side to thirteen on the right.

The clinical disturbance wrought by them is variable. No symptoms may be produced, their existence being accidental discoveries

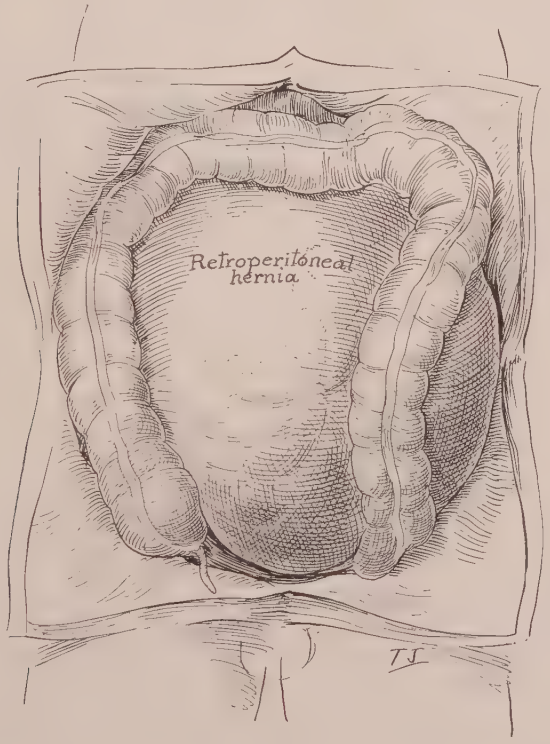

Fig. 88.—Retroperitoneal hernia displacing the colon. (Redrawn from Neubauer.)

of the autopsy room. Chronic disturbances are caused, even to the more violent symptoms of acute obstruction.

When a considerable mass of intestine escapes behind the peritoneum a more or less circumscribed tumor is produced. It is the discovery of a tympanitic tumor which, according to Leichtenstern, sometimes makes it possible to make a provisional diagnosis. According to him when a circumscribed globular distention, tympanitic on percussion, a firm, elastic lump, giving the impression of

a slightly movable cyst, is found, a retroperitoneal hernia may be suspected. The presence of hemorrhoids, suggesting pressure on the inferior mesenteric veins and digestive disturbances, increases the probability according to him. I once made a diagnosis on such findings, and at operation a localized tuberculous peritonitis was found.

Hernia into the Sigmoid Fossa.—But two cases of this type of fossa have been recorded. Eve and Eccles have contributed the cases. In Eve's case a six-inch loop was strangulated, and the patient died of acute obstruction. In Eccles' case four inches of bowel was imprisoned, and the patient died after resection.

Hernia into the Foramen of Winslow.—Moynihan accepts eight of the recorded cases as genuine. The diagnosis, when the field of disease is laid bare, should be easy. An elastic tympanitic epigastric tumor, together with symptoms of acute obstruction, seem to be the factors which would lead one to suspect such a condition.

Genesis of Retroperitoneal Hernias.—The mechanics of the formation of the retroperitoneal hernias must be different from that operative in other hernias. In ordinary hernias the escape of gut is due to the fact that the pressure within the abdomen is greater than that in the hernial sac, existent or potential. In the case of retroperitoneal hernias where the entire small gut is retroperitoneal, it is impossible to believe that the pressure within the abdomen is greater than that in the huge sac containing the gut.

The study of a case recently reported by Twyman makes me believe that another explanation is more plausible. This case presented the folding over of a part of the small intestine by the ascending colon and its fixation there (Fig. 89). The resemblance of this condition to some of the reported cases, for instance, Broesike's (see Moynihan, p. 60, Fig. 19) makes it seem that this condition is but an exaggeration of that condition observed in Twyman's case. In other words, it seems possible that right duodenal hernias are caused by the covering over of the intestinal coils by the meson of the ascending colon, which afterwards becomes fixed, thus forming a permanent housing of the small intestines (Fig. 90). In early embryos one occasionally finds the cecum and its mesentery floating on the small intestines. Usually,

of course, the cecum may continue to float but the possibility of its becoming attached does not seem to place such a heavy tax on the imagination as the assumption that the gut becomes retroperitoneal because of the increased intraabdominal pressure

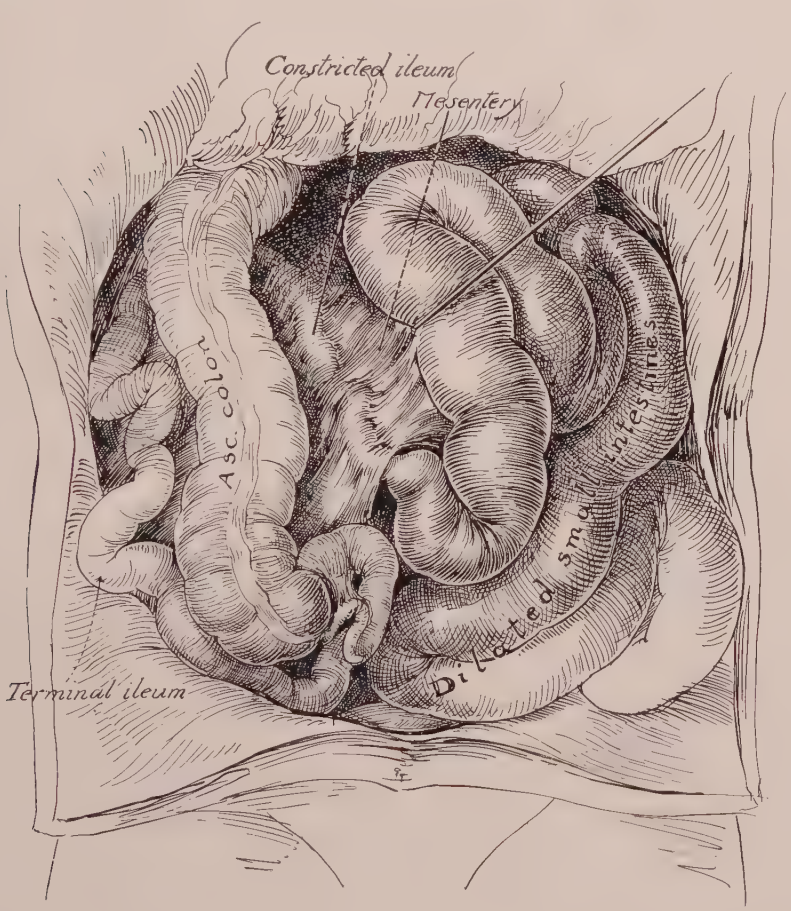

Fig. 89.—Retrocolonic position of a portion of the ileum. (Twyman's case.)

When we seek to explain the left duodenal hernias on the same basis greater difficulty is encountered. I have noted in young embryos that sometimes the inferior mesenteric vessels are very prominent, forming a veritable pocket with this vessel forming

a rigid rim. If the splenic flexure did not tend to obliterate this space by seeking the splenic region, this cavity might easily become enlarged. As a matter of fact in the recorded cases of duodenal hernias the splenic flexure of the colon did not occupy its usual site, but lay on the top of the sac just as this hypothesis would demand. This is exemplified by Neubauer's case. (Moynihan, p. 52.) This point never having been considered, the records of course are

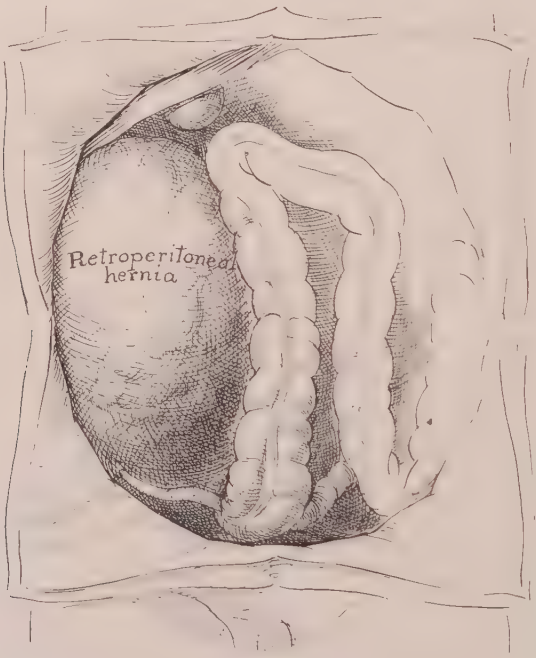

Fig. 90.—Right retroperitoneal hernia. (Redrawn from Brösike.)

faulty on this point. Perhaps future operations may add further light.

The Omentum.—The name omentum was first used by Celsus. The origin of the term is not fully established. One explanation is that it is derived from operimentum, signifying that it covers something (operit), namely, the intestines. Huschke believes the word was derived from omen, because the Haruspices made prognostications from its structure. The one favored by Hyrtl (the

foremost anatomic linguist) is that it is derived from opimus = fat. The Arabic equivalent is zirous = fat. The origin of the name is of interest because it reflects the opinion of these early writers regarding its function.

Development.—The development of the great omentum has been sufficiently considered in the section on the formation of the bursa omentalis. It remains now only to trace its late development, and to consider its final relations with the organs with which it comes in contact. If its development is kept in mind the anomalies of its form are easily understood. Koch has collected the cases of anomalies reported up to that time. These consist of a marked abbreviation in its length or to its partial or complete failure to unite with the transverse colon. Its abbreviated length is easily explained by a failure of initial development. The extent to which this may be observed is illustrated in a case recorded by Schiefferdecker, in which the great omentum was represented by a cicatricial ridge extending along the lower border of the colon. A more common anomaly is the failure of the omentum to unite with the transverse colon. This anomaly, it is easy to understand, may readily be produced by a failure of attachment as the organ grows over the colon. Kirschner reports an example of this anomaly. The case of Kirschner's is of philogenetic interest, since the defects appeared as a result of the failure of the splenic flexure to rotate, retaining what, in certain anthropoids, is a normal state.

Partial failure to unite is not uncommon, and union beyond the usual confines is likewise often observed. The latter anomalies are often charged to fetal or postnatal peritonitis.

Extent and Position of the Great Omentum.—The omentum extends from the transverse colon, as far as the symphysis pubis below and to or over the colon on either side (Fig. 91). This ideal position is often noted at the operating table when the abdomen is opened for noninflammatory lesions. In the presence of inflammatory lesions, or in abdomens previously the site of reactive processes the location may be much altered, depending in degree upon the site, character, and extent of the former lesion.

The attachment of the great omentum to the colon extends from the gastrocolic ligament to the second portion of the duodenum. The greater extent of its attachment is along the greater curva-

ture of the stomach. As it extends over the transverse colon it is attached to the upper anterior border of this gut. To the left

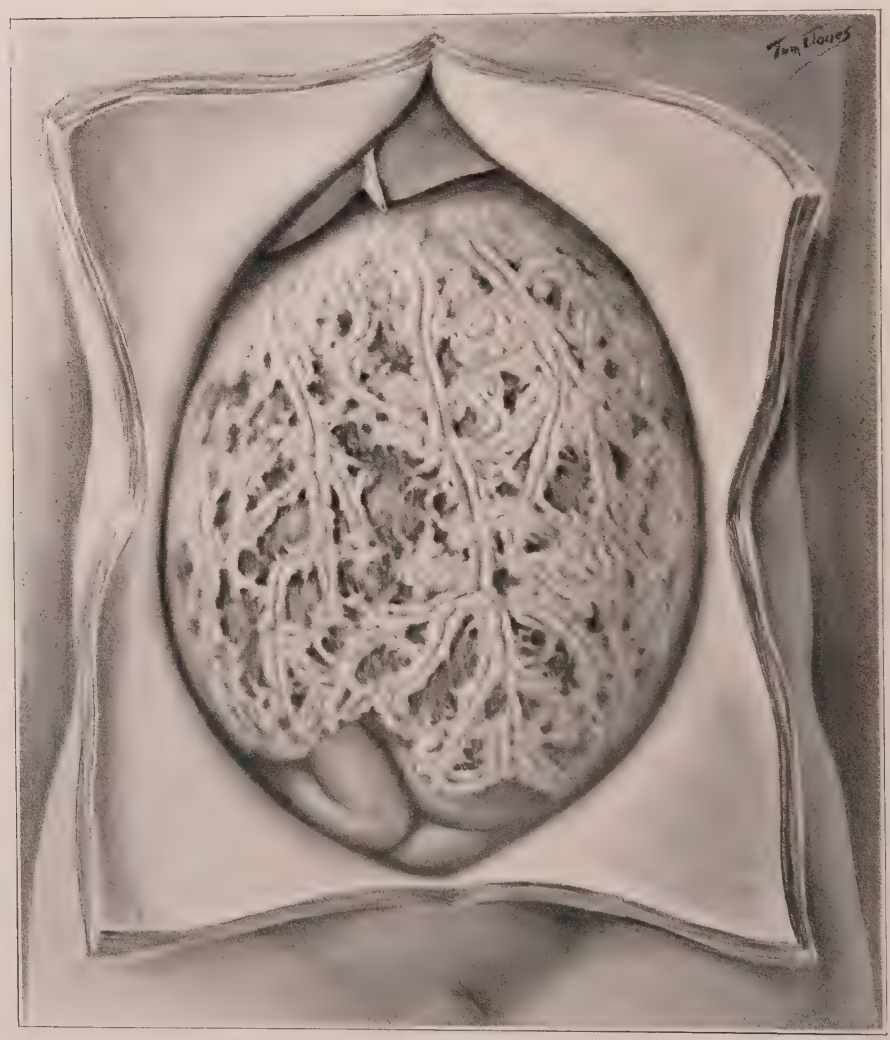

Fig. 91.—The normal great omentum.

it is attached to the gastrosplenic and phrenocolic ligaments. From this point the lower plane is continuous with the peritoneum over the body of the pancreas, extending to the right on the second

portion of the duodenum. At the right it is continuous with the hepatic duodenal ligament and in a considerable portion of cases with the gall bladder itself, forming a meson for it. To the right it terminates as a falciform ligament, extending from the hepatoduodenal ligament to the hepatic flexure of the colon. The omentum is not infrequently attached to the ascending colon. This condition was regarded by von Haller as a normal variation. Some modern surgeons believe it pathologic. In infrequent instances the omentum may attach to the lateral abdominal wall, also regarded by von Haller as within the normal. Sometimes the right portion of the omentum is separated from the main portion, and is then called an accessory omentum.

The relation of the omentum to the gall bladder is a matter of special interest, since this union is often regarded as being of inflammatory origin. This close association of the gall bladder and omentum should not occasion surprise, for, when the omentum first becomes attached over the colon, it comes into direct contact with the gall bladder at a time when both these structures are in a very plastic state.

In the dead-house the ideal location of the omentum is seen only in bodies dead of accident or acute extraabdominal pathologic processes.

In chronic diseases, even in the absence of specific intraperitoneal lesions, the location of the omentum is exceedingly varied. It is often found rolled up beneath the colon, about the stomach, between the stomach and liver, or even between the liver and diaphragm. The unusual locations are most frequently noted in bodies dead of chronic exhaustive lesions attended by great emaciation. The cause of these peculiar migrations is discussed in a subsequent chapter.

As already noted in the chapter on development, the omentum is developed from the primary peritoneal plates. In its ultimate form there is nothing to remind one of this complicated genesis. It is made up of a network of large vessels running at some distance from each other. The intervascular areas present a thin transparent membrane, formed of two peritoneal plates with a connective-tissue network bearing potential vessels. In some instances these intervascular planes become perforated. The ves-

sel-bearing connective-tissue bundles with openings between these interdigitations form a veritable network, fully justifying the term "Netz" applied by the Germans (Fig. 92).

The cause of the fenestration which the great omentum sometimes suffers is not known. Zorner advanced the hypothesis that it is due to the rapid development of the organ. His idea was that the vascular areas grew so rapidly that the thinner intermediary spaces could not keep up, and, as a consequence, they became so attenuated that a solution of continuity took place. This theory seems plausible, for fenestration is most often noted

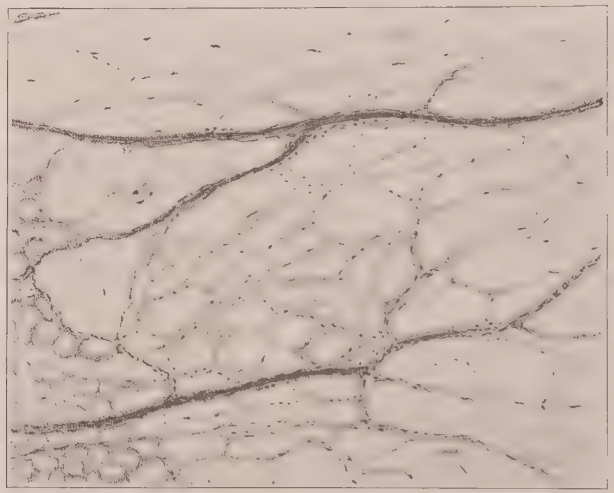

Fig. 92.—Low magnification of a segment of omentum showing the small fenestra between the vessels.

in fat omenta. It is easily conceivable that an enormous development of fat might make extra demands on the peritoneal enveloping surface; and the thinner avascular fields may readily be conceived of as being the areas most likely to suffer. Ranvier's theory lays the blame on the leucocytes. He believes that leucocytes in the course of their migration may perforate first one surface and then the other, ultimately leading to a solution of continuity of both layers. An opening once made may readily be enlarged. It is difficult to confirm or disapprove either of these theories. The avascular fields of the omentum contain much less

connective tissue than do similar areas in the mesentery, and in consequence are much more subject to injury. Many omenta apparently not fenestrated when examined with the naked eye are seen to be so when examined microscopically.

The thickness of the omentum as a whole is subject to enormous variations. In emaciated persons the entire omentum may not weigh more than a few ounces, while in others the omentum is as much as three or four inches thick, or even more, extending over the entire abdomen and weighing in the aggregate as much as fifteen pounds.

Blood Vessels.—There is but little in the anatomies relative to the blood vessels of the omentum. Merkel states that the great omentum is supplied by six or eight long, thin arteries which arise from the gastroepiploric vessels, and course in the anterior layer. The vessels are not easily demonstrated unless the organ is in a state of reaction. They seem for the most part to be potential vessels, as described for the mesentery. They accompany the fibrous bundles of which the network is composed. When irritated they may become vessels easily recognized by the naked eye. This is particularly true when it supplies permanently a crippled tumor.

The veins are larger, and are readily recognized. When in a state of reaction, as in supplying a tumor, they may be as large as a lead pencil or larger.

When dilatation due to reaction is prolonged it may become permanent, and a state of varicosity may remain. In the adult omentum quite commonly areas of large veins may be observed, being the result of reactive or passive stasis. It is difficult on this account to form a picture of the normal.

Lymph Vessels.—The information available regarding the lymph vessels is very indefinite. Sappey, one of our foremost authorities on the anatomy of the lymphatics, denies their existence in the great omentum entirely. Luschka notes that the great omentum contains a few lymphatics, which communicate with the lymph glands at the greater curvature of the stomach. Norris made a most careful study. He divides the lymphatics into two categories,—a fine superficial and a large deep plexus. Only the former follow definitely the course of the vessels. Suzuki defined the location of lymphatics by sectioning portions of the organ after rup-

ture of the liver in carcinoma of the abdominal organs. When he discovered clumps of cells in his sections he concluded that they occupied the lymphatics. Having noted the facility with which cells become encapsulated in the great omentum, it seems to me that conclusions based on these findings must be accepted with some reserve.

Neural Anatomy of the Peritoneum.—As the peritoneum is primarily a protecting organ, it must be assumed that it possesses a nerve supply quite in harmony with its function. The parietal peritoneum supplied by cerebrospinal nerves, acts with the abdominal wall as a protecting organ against insults from external forces. It possesses, in addition to the sensory nerves, sympathetic fibers associated with those of the visceral layer. A sensory system is thus provided which warns the organism of impending danger, while the parietal mechanism is concerned chiefly in warning the animal of dangers from without. The visceral nervous mechanism must give evidence of abnormal distention and dislocation of the internal organs. Because of this difference in function of the nerve supply of the parietal and the visceral layers it is necessary to treat them separately.

Parietal.—Historical: Haller was the earliest writer to speak of the nerve supply of the peritoneum. He believed the peritoneum was devoid of nerves; he thought that those lying near that structure belonged to the muscles. Bajard likewise was unable to trace nerves to the peritoneum, as was Reichert. Bourgery commented on the assumption of other anatomists that the peritoneum was devoid of nerves, and he declared that, quite to the contrary the peritoneum is very abundantly supplied with nerves. His greatest success in demonstrating the presence of nerves was evidently due to his improved technic. He macerated the tissue in 1 to 2 per cent nitric acid and examined them under three to ten power magnification.

Luschka declared that the peritoneum derives its nerves from the phrenic and intercostal nerves. He laid particular stress on the division of the phrenic, which he concluded was distributed in the general direction of the umbilicus. This distribution of the phrenic in the anterior abdominal wall, he believed, accounted for the greater pain in the region of the umbilicus in peritoneal

inflammations. He makes no further mention of the intercostal and lumbar nerves other than to say that they supply the peritoneum. Most writers have followed Luschka in emphasizing the distribution of the phrenic to the exclusion of the other nerves.

Cyon studying the cisterna magna lymphatica of amphibians by means of the gold-chloride method was able to demonstrate medullated fibers which terminated in delicate end-organs. Jullian using the same method was the first to definitely describe the end-organs. He stated that they ended in little enlargements which were ovoid, or pear-shaped. He also described nerves in the great omentum. Finkam described nerves in the human omentum. He doubted if Jullian had actually seen end-organs.

The Intercostal Nerves.—The parietal peritoneum receives branches from the lower six thoracic nerves and from the ileoinguinal and the ileohypogastric. The thoracic nerves pass forward after leaving the spinal canal to the posterior surface of the intercostal membrane, which they pierce, and pass forward in company with the intercostal vessels between the intercostal muscles. At the costochondral junction they divide after passing behind the costal cartilages, giving off branches, with which we are not concerned here, to supply the superficial layers of muscles and the skin and numerous smaller branches which supply the transversalis, the rectus and the underlying peritoneum in which we are chiefly interested.

These nerves near the costochondral junction divide into numerous branches, which pass forward divergent into or upon the transversalis. It is these nerve branches which supply the nerves to the parietal peritoneum, and which are, therefore, deserving of careful study, not only because of the theoretical interest, but because it is there that the common origin is found. This is a matter of interest in local anesthesia, and because of their relations here many of the referred pains are found.

The nerves proceed medially either within or upon the transversalis muscle, forming plexuses by the reunion of branches of the same nerve and by the inosculation with branches of neighboring nerves. The greater number of anastomoses take place within the substance of the muscle, and it is from these loops that the filaments are given off which supply this muscle itself.

It is from these loops, too, that the nerves to the peritoneum are given off. Some of them pass at once from the loops from which they arise directly through the substance of the muscle, through the fascia into the peritoneum. Others run for some distance within or upon the substance of the muscle toward the semilunar line, and, either before or after passing this line, pass through the fascia to the peritoneum. Others pass more medially over the epigastric vessels before reaching the peritoneum.

Within the subperitoneal tissue or within the peritoneum itself the nerves undergo renewed plexus formation, and may assume a recurrent course toward the point of origin before breaking up in their end-organs. On the whole the general direction of the terminal branches is toward the median line. In addition, in harmony with the developmental changes of position of the abdominal muscles, the terminal filaments are directed upward. In this deviation they follow the general course of the nerves supplying the overlying muscles.

It is because of this general harmony of direction of the peritoneal and muscle branches that a more careful study of the overlying muscle supply is imperative. In the early embryo the arrangement is simple. Here the nerves pass directly from their origin to the midline, and it is on account of the progressive rotundity of the abdomen, demanding an extension of the abdominal muscles, that the nerves supplying the abdominal wall are deviated from their primitive course. It is only by following the nerves from their point of origin that the relation of the nerve root to the muscle segment can be determined, though it is safe, following developmental laws, to assume such relationship. Nevertheless, it is comforting to have the assurance of actual demonstration. Each muscle segment receives its nerve supply from a definite nerve root, but with free anastomosis with neighboring nerves, particularly from the nerves lying next caudad, that is to say, the chief direction of the branching is cephalad.

When the nerves approach the rectus they divide at its lateral border into two groups, the one lying lateralward and the other medial to the epigastric and inferior mammary vessels. They divide and reunite forming plexuses within the substance of the muscle but retain an intimate relationship with the group on the

other side of the vessel above named by a free network of fibers.

The same general distribution is maintained by the lower abdominal nerves,—the iliohypogastric and the ilioinguinal. The more divergent direction of these lower nerves is due to the greater expansion of the myotomes which these nerves accompany. The general tendency of the branches of the nerves to curve cephalad is emphasized by the hypogastric branch of the iliohypogastric (Bardeen and Lewis).

Ramstrom has studied particularly the relation of the intercostal nerves to the diaphragm and the phrenic to the peritoneum. He denies the correctness of the general teaching that the diaphragm receives motor fibers from the intercostal nerves and, conversely, that the peritoneum receives fibers from the phrenic. He traced twigs from the intercostal nerves at the diaphragm insertion into the parietal peritoneum and into the peritoneum of the diaphragm, but not in the muscles of the diaphragm. In the region where the peritoneum was believed to receive fibers from the phrenic nerve he demonstrated fibers from the sixth and seventh intercostal nerves. He was unable to follow any fibers from the phrenic beyond the diaphragm. Numerous dissections of my own cause me to accept with confidence these conclusions.

The sensory nerves of the pleural surface of the diaphragm have been studied by Capps. By irritating various areas of the diaphragm through a cannula, passed for the purpose of draining off effusions, he came to the following conclusions: The outer rim of two or three inches receives its supply from the intercostal nerves, while the central portion receives its supply from the phrenic. He comes to this conclusion because of the referred pain. Arguing in the same manner for the abdominal side of the diaphragm, we may conclude that the whole of this surface is supplied by branches from the intercostal nerves, since there are no neck pains when this side of the diaphragm alone is inflamed.

Timofejew agrees that the phrenic does not supply the parietal peritoneum, but finds branches reaching the diaphragm through the hepatic ligaments (Eisler).

The terminals of the nerves of the peritoneum have been studied, particularly by Dogiel. The fibers which are given off from the plexuses above mentioned, according to him, are nonmedullated for

the most part, and a small number of variously sized medullated fibers. The latter divide at the nodes of Ranvier into numerous branches, so that medullated fibers are found in most meshes.

The nonmedullated end in fine meshes about the blood vessels. Sometimes in the course of the nerves forming the meshes, little groups of sympathetic nerve cells, or single cells, are found. The medullated fibers end in the serous and subserous layer in special end-apparatus. These end-organs may be divided into two groups, the encapsulated and the nonencapsulated.

Encapsulated End-Organs.—These are of the type of Vater-Pacini bodies, as seen in the tendinomuscular junctions and in the skin. They differ from the latter in size and form, only being for the most part elongated, blunt-ended cylinders, but some are globular or egg-shaped. Some of these end-organs lie very superficial, almost beneath the endothelium. Here they lie parallel with the surface. On the whole the number of end-organs is not great. In some areas of 37 to 45 sq. cm., Dogiel found 45 to 53, that is, roughly speaking, an average of 1 per sq. cm. They are most numerous in the anterior abdominal wall near the median line. According to this author they lie deeply imbedded in the subperitoneal connective tissue. The exact relation to the peritoneum or subperitoneal tissue is conjectural. Because of the staining technic employed it is difficult to determine their exact position. No one, so far as I know, has ever been able to demonstrate them in cross sections. These end-organs usually occur in groups of two to four. Thus, large areas occur without the presence of end-organs. The technic in demonstrating them is uncertain, and it is likely that, even in the most successful specimens, many remain invisible because unstained.

These end-organs vary in size from 0.025 to 0.193 mm. by 0.012 to 0.043 mm. Timofejew describes them as measuring 240 to 260 microns in length and from 60 to 100 microns in breadth, the larger ones being curved or S-shaped. He has observed still larger and more slender ones in the rabbit measuring 300 to 500 microns in length and but 25 to 30 microns in breadth. Like the Vater-Pacini bodies, they are formed by numerous lamellar cells, between which are connective-tissue cells with prominent nuclei. Timofejew describes the lamellæ as being composed of from 5 to 6 layers. In

addition there is a fine capillary network lying close upon the outer surface.

The medullated fibers which terminate in these end-organs are characterized by their thickness and the great distance between the nodes. They may branch many times before terminating in the end-organs. They lose their sheath just before disappearing in these organs, entering them as naked cylinders. After entering in the end-organs they break up into numerous filaments, which describe a serpentine course as they approach the termination of the end-organs. In their course these filaments give off numerous small lateral branches.

In many specimens the medullated fibers after branching from the primary meshwork are accompanied by nonmedullated fibers and their medullated fibers. The nonmedullated fibers approach the end-organs. They are arranged about the capillary network above mentioned. The accompanying fine medullated fibrils lose their sheaths at some distance from the end-organs, and enter with the larger medullated fibers already described. After entering they divide into fine fibrils, which are distributed about the periphery of the cavity of the end-organs.

Nonencapsulated.—These are of two types, the one located just beneath the endothelium, the other situated more deeply in the subperitoneal tissue. In the meshwork above mentioned numerous small branches are given off and pass out like roots in many directions. From these are formed from ten to seventeen medullated fibrils which after extending for some distance suddenly lose their sheath and neurilemma while the axis cylinder divides into two or three branches. These again divide into a large number of short branches, which undergo leaf-like expansion before they terminate.

The nonencapsulated end-organs, though less conspicuous and striking, are vastly more numerous, and are regularly arranged.

The diaphragmatic peritoneum is supplied from the phrenic, the intercostal and the sympathetic nerves, which reach it by way of the hepatic ligaments.

The Phrenic Nerve.—After reaching the diaphragm the phrenic nerve passes into it and ramifies on the abdominal side, forming an abundant plexus. This is added to by the nonmedullated nerves

which reach the diaphragm by way of the hepatic ligaments. The intercostal nerves reach the diaphragm at the point of interdigitation of the diaphragmatic with the parietal muscles.

At the point of junction of the plexus formed on the serous membranes are seen small sympathetic ganglia made up of five to ten multipolar cells. These nodes are most numerous at the centrum tendineum.

Timofejew regards the nonmedullated fibers as vasomotor because they can be traced along vessels for long distances. The medullated fibers from the phrenic and intercostals are motor and sensory, since fibers can be traced to the muscle cells. In both the diaphragmatic and pleural peritoneum, they terminate in typical end-organs. The sensory nerves end in the subperitoneal tissue, either as free tufts or as encapsulated end-organs, the latter being less numerous than the former, and situated for the most part in the centrum tendineum.

External Topography of the Abdomen.*—The anatomist is not concerned with the division of the abdominal cavity into regions; but the clinician finds such a division of great practical convenience, for he has learned that symptoms in a certain region mean a disturbance of organs lying at or near such regions.

The time-honored custom of dividing the abdomen into quadrangles is a purely arbitrary one without rhyme or reason. It totally disregards the relation of abdominal contents. By using certain bony prominences as starting-points, there result certain squares, which remind one of the scheme for quartering beeves as exhibited in the old family almanac. The belief that such division was without value caused me early to seek for a division which should keep in mind the abdominal contents. This search was pursued by studying the abdominal contents devoid of the covering muscles. Regions were mapped out which should represent, as nearly as possible, disease entities or disease groups. In considering such regions, not only the organs they contain, but particularly the location of the symptoms expressive of disease of these organs should be taken into account. Certain organs which because

*This account is taken with modifications, from a paper published by me in the St. Louis Courier of Medicine, 1904, xxx, 1-8.

of their attenuated form are not confined to any region, lend themselves poorly to such a division.

After such division has been made the bony structures were examined for possible fixed points for the projection of the lines. There results a series of triangles, which in a fairly accurate manner includes organs and symptom points (Fig. 93). The umbilicus

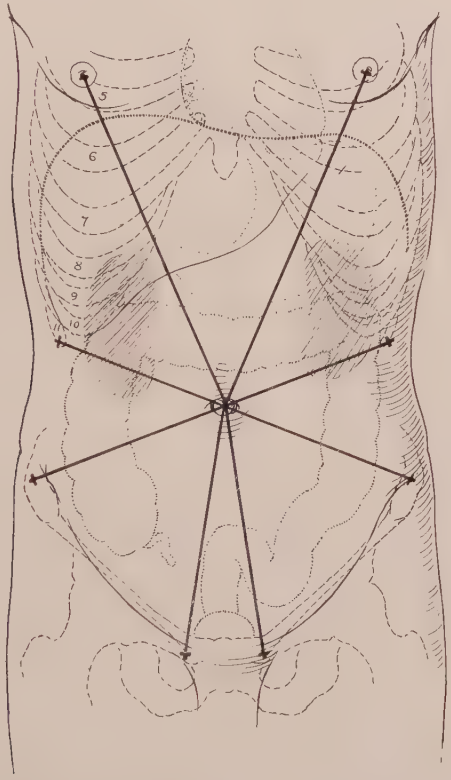

Fig. 93.—Author's division of the surface of the abdomen into triangles.

is used as a center from which radiating lines are drawn, resulting in a series of relatively equiangular triangles.

The first of these triangles is included by two lines beginning at the umbilicus and radiating to the eighth chondral tip or to the nipple on either side. There results a triangle which includes the major portion of the stomach, and is the site of the

clinical phenomena arising from that organ. Pain and tumors referable to this organ find expression in this triangle, at least in their incipiency. This region may fittingly be called the gastric or epigastric triangle.

A line projected on the right from the umbilicus to the tip of the eleventh rib takes in the region occupied by the gall bladder and bile ducts. The pains experienced from disease of this organ find expression in this region. If planes be passed in the direction of the lines bounding this triangle they will define the region of referred pain in the back. The enlarged gall bladder presents in this triangle, and it is only when excessively large or mobile that it extends beyond its confines. This triangle may be called the cholecystic or hepatic region.

The next line is drawn from the umbilicus to the tubercle on the crest of the ileum. It passes below the right kidney and about 5 cm. above the ileocecal junction. I have selected the tubercle, instead of the anterior superior spine, in order that it may pass well above the ileocecal junction. Since this triangle contains nothing of importance, except the kidney and its capsule, it may be properly designated the renal region.

The next line may be drawn from the umbilicus to the tubercle of the pubis. The tubercle is usually located 3 or 4 cm. lateral to the midline. This triangle takes in the appendix, the ileocecal junction and the structures involved in inguinal hernia, and may therefore be called the right ileocecal or ilioinguinal region.

The next line extends to the left tuberculum pubicum, and encloses the region of the uterus and bladder, and may be called the vesicouterine region.

The next line extends from the umbilicus to the tubercle on the crest of the ileum, corresponding to the line on the right side. It contains the sigmoid, and may be called the sigmoid region, or because of the importance of the inguinal structures it may be called the left ilioinguinal region.

The next line extends from the umbilicus to the tip of the eleventh rib on the left side. This triangle contains the kidney and its capsule, and is, therefore, the left renal region. This line extended follows nearly parallel with the eleventh rib, and separates, as accurately as possible, the spleen from the kidney.

The remaining triangle is separated from the epigastric, already mentioned, by a line extending from the umbilicus to the eighth costochondral junction. It contains the spleen, and is at the point where this organ is always searched for by palpation. When this organ enlarges it appears first in this region. Obviously, it must be called the splenic region.

Some of these regions admit of secondary divisions. This is true of the ileocecal region. This region, as already described, is practically an equilateral triangle. It may be subdivided into three

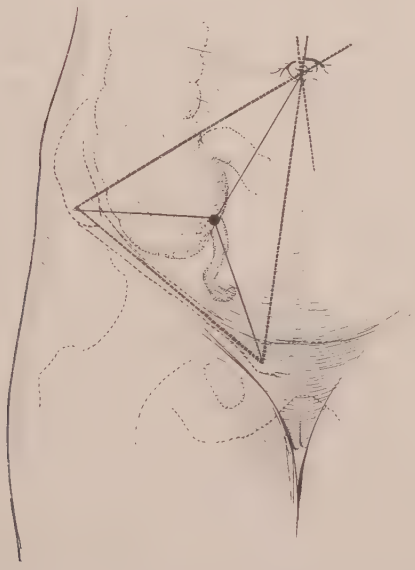

Fig. 94.—Secondary division of the ileocecal triangle into secondary triangles, the common apex of which corresponds to the base of the appendix.

equilateral triangles. The common apex of these triangles corresponds to the base of the appendix (Fig. 94). The studies on which this statement is based were made by laying off the triangles on the abdomen of a cadaver, and then pushing a long steel pin through the points indicated, and fixing the pin by driving it into the bone beneath. The abdomen was then opened and the relation of the pin to the base of the appendix noted. In fully 80 per cent of several hundred cases studied the pin placed at

the point indicated penetrated within a centimeter of the base of the appendix.

McBurney's point has reference to the location of maximum pain in appendicitis. His conclusions are based entirely on clinical observations, and make no pretense at anatomic exactness. The usual

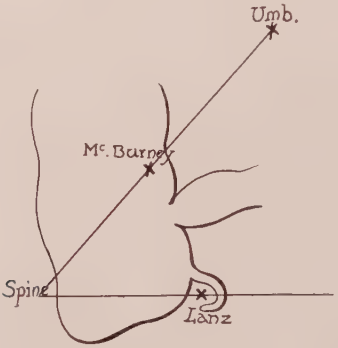

Fig. 95.—McBurney's point compared to Lanz's point.

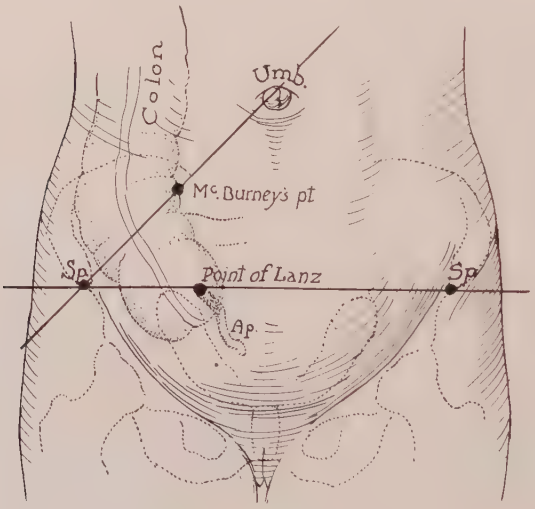

Fig. 96.—Lanz's point.

understanding is that his point is located midway between the umbilicus and the anterior superior spine of the ileum. McBurney's own words are, "I believe that in every case the seat of greatest pain, determined by the pressure of one finger, has been very ex-

actly between an inch and a half and two inches from the anterior superior spinous process of the ilium on a straight line drawn from that process to the umbilicus." (Fig. 95.)

Lanz also attempted to define the location of the appendix more accurately. He drew a line from one anterior superior spine to the other and defined as his point the junction of the right and middle thirds of this line (Fig. 96). In his studies of 20 cases he found the appendix to lie lateral to this point and it lay exactly beneath it in only two cases. In the remainder it lay either above or below this point. According to his studies, likewise, my point corresponds more nearly to the site of the base of the appendix. His scheme has the merit of the greater simplicity. Garau found Lanz's point to correspond to the site of the appendix in 86 per cent of the bodies examined. He believes that McBurney's point corresponds to the area of distribution of the spinal nerves which are united with the afferent nerves from the appendix by the spinal ganglia.

The hepatic triangle, likewise, may be subdivided. If this triangle is divided into three equal triangles the common apex will correspond with the location of the cystic duct.

Bibliography

Bajard: Dissertation sur sérieuse phlegmasus des membranes serieuses, Paris, 1818.

Bardeen and Lewis: The Development of the Limbs, Body-Wall and Back. Am. Jour. Anat., 1901-2, i, 1.

Bardeen: Growth and Histogenesis of the Cerebro-Spinal Nerves in Mammals. Am. Jour. Anat., 1902-03, ii, 231.

Bartels: Das Lymphgefässysteme, Fischer, Jena, 1909.

Bayer: Entwicklungsgeschichte und Anatomie des weiblichte und Anatomie des weiblichte des weiblichen Genital-apparates, Strassburg, Schlesier und Schweikhardt, 1908.

Berger: Hernies inguinales Varietés rares., In Duplay and Reclus, Traité de Chirurgie, ed 2. Masson et cie: Paris, 1898, vi, 208.

Berry: The Cæcal Folds and Fossæ, William F. Clay, Edinburgh, 1897.

Blaisdell: The Anatomy of the Sacro-uterine Ligaments, Anat. Rec., 1917, xii, 1.

Bockdalek: Uber den Peritonealüberzug der Milz und das Ligamentum pleurocolicum, Arch. f. Anat. u. wissensch. Med., 1867, 565.

Bourgery: Mémoire sur les nerfs des membranes séruses en général. et sur ceux du peritoine en particulier chez l'homme, Compt. rend. Acad. d. sc., Paris, 1845, xxi, 56.

Broesike: Ueber intraabdominale (retroperitoneale) Hernien und Bauchfelltaschen, nebst einer Darstellung der Entwicklung peritonealer Formationen, Berlin, Fischer, 1891.

Brügel: Ein Fall von Pyämie im Anschluss an einen subphrenischen Abszess bei chronischer Cholelithiasis, Erlangen, 1902, No. 23.

Capps: An Experimental Study of the Pain Sense in the Pleural Membranes, Arch. Int. Med., 1911, viii, 717.

Charpy: La gaine des muscles deposits et le cavaté prévésicale; In; Études d'anatomie applequée, Paris, Bailliere, 1892, p. 183.

Clado: Appendice caecal; anatomie, embryologic anatomie comparée bacteriologie normale et pathologique., Compt. rend. Soc. de biol., 1892, 9 S., iv, pt. 2, 133.

Cyon: Ueber die nerven des Peritoneum, Arb. a. d. physiol. Anst. z. Leipzig, 1896, iii, 104.

Dailey: Total Congenital Absence of the Vermiform Appendix, Surg. Gynec. and Obst., 1901, xi, 413.

Debierre: Traité élémentaire d'anatomie de l'Homme, Paris, Felix Alcan, 1890.

Dogiel: Arch. f. mikr. Anat., Bonn, 1902, lix, 1.

Durand: Le ligament ilio-ovarien (appendiculo-ovarien de Clado) contribution à l'étude des ligament large, Progrès méd., 1895, 3 S., ii, 1.

Dwight, F. B.: Piersols Anatomy, Philadelphia, J. B. Lippincott Co., 1908.

Eastman: The Foetal Peritoneal Folds of Jonnesco, etc., Surg., Gynec. and Obst., 1913, xvi, 341.

Eccles: A Case of Strangulation of a Loop of Small Intestine in the Fossa Intersigmoidea, St. Barth. Hosp. Rep., London, 1895, xxxi, 177.

Eisler: Verhandl. d. anat. Gesellsch., Jena, 1902, S., 202-207.

Eliot: A Report of the Unusual Cases of Appendicitis, Med. and Surg. Rep. Presbyterian Hosp., N. Y., iii, 173.

Engel: Einige Bemerkungen über Lageverhältnisse der Baucheingeweide in geuenden Zustande, Wien, med. Wchnschr., 1857, vii, 553, 585, 601, 641, 673, 705, 737.

Eppinger: Hernia Retroperitonealis, Vrtljschr. f. d. prakt. Hielk., 1870, cviii, 121.

Eve: A Case of Strangulated Hernia into the Fossa Intersigmoidea, Brit. Med. Jour., 1885, i, 1195.

Farra-Todd: Cyclopedia of Anatomy and Physiology, London, Longmans, Greene & Co., 1859, suppl. v, p. 550.

Ferguson: Some Important Points Regarding the Appendix Vermiformis, Am. Jour. Med. Sc., 1891, cl, 61.

Finkam: Ueber die Nevenendigung des groszen Netze, Gottingen, 1873.

Flint: Embryonic Bands and Membranes about the Cecum, Bull. Johns Hopkins Hosp., 1912, xxiii, 302.

Fowler: Observations upon Appendicitis, Ann. Surg., 1894, xix, 14.

Garau: Le point de McBurney et le point de Lanz, Lyon chirurg., 1910, iii, 485.

Gerota: Beitrage zur Kenntniss des Befestigungsapparates des Niere, Arch. f. Anat. u. Entwicklungsgesch., 1895, p. 266.

Gruber: Bildungshemung der Mesenterium, Arch. f. Anat. u. Physol., 1862 and 1864.

Haen, de: Ratio medendi in nosocomio practico, Parisiis, Didot, 1769, vi, 103.

Hasselbach: Die Erkenntniss und Behandlung der Eingeweidebruche durch naturgetreue Abbilgungen erläutert Nürnburg, Bauer and Raspe, 1840.

Haller, v.: Elementa, physiologiae corporis humani, Lousanne, Berne, 1766, vii.

Hartmann: Die Bauchfelltaschen in der Umgbung des Blinddarmes, Tübingen, H. Laupp, 1870.

Henke: Topographische Anatomie des Menschen in Abbildung und Beschreibungen, Berlin, Hirschwald, 1884.

Der Raum Bauchhöhle des Menschen, und die Vertheilung der Eingeweide in demselben, Arch. f. Anat. u. Entwcklngsgesch, 1891, 89.

HENLE: Handbuch der systematischen Anatomie des Menschen, Vieweg, Braunschweig, 1866, ii.

HENSING: Die peritonæo giesæ, 1742, In; Haller Dis. Anat. (etc.) Gottingæ, 1750, i, 349.

HERTZLER: The Treatment of Inguinal Hernias in Children, Jour. Am. Med. Assn., 1913, lxi, 1879.

Operation for Shortening of the Broad Ligaments, Jour. Am. Med. Ass., 1907, xlviii, 277.

HESSERT: The Frequency of Congenital Sacs in Oblique Inguinal Hernia, Surg., Gynec. and Obst., 1910, x, 252.

HIBBEN: Guy's Hosp. Reports, 1867, p. 21.

HILDEBRAND: Grundriss der chirurgisch-topographischen Anatomie, ed. 2, Wiesbaden, Bergmann, 1900, p. 232.

HUNTINGTON: Anatomy of the Peritoneum and Abdominal Cavity, Lea Bros., Philadelphia, 1903.

HUSCHKE AND HENLE: Lehre von den Eingeweiden und Sennesorganen des Menschlichen Korpers, In v. Soemmering's Handbuch, Leipzig, L. Voss., 1844, v. 5.

Handbuch der topographischen anatomie, ed. 7, Wien, Braumüller, 1882.

HYRTL: Handbuch der topographischen anatomie, ed 7, Wien, Braumüller, 1882, p. 758.

JACKSON: Membranous Pericolitis and Allied Conditions of the Ileocecal Region, Ann. Surg., 1913, lvii, 375.

JONNESCO: Hernies internes rétro-péritonealés, Paris, Steinheil, 1890.

JULLIEN: Contributions a l'étude du peritoine; ses nerfs et leurs terminaisons, Lyon méd., 1872, xi, 371; 380.

KELYNACK: Pathology of the Vermiform Appendix, London, Lewis, 1893.

KIRSCHNER: Ueber einen Fall mesenterialer Bildungsanomalie, Beitr. z. klin. Chir., 1909, lxi, 615.

KOCH: Arbeiten aus der chirurgischen Universetats Klinik Dorpat, 1903, Heft. 5.

KÖNIG: Ueber die Bedeutung der Spalträume des Bindegewebes für Ausarbeitung der entzündlichen Processe, Samml. klin. Vortr., 1872, No. 57 (Chir. No. 18,359).

KRAUSE: Handbuch d. Anat., 1876, i, 298.

LACENET: Mém. Acad. de St. Petersburg, Series vii, xxviii, 181.

LANDZERT: Über die Hernia retroperitonealis (Treitz) und ihre Beziehungen zur Fossa duodenojejunalis, St. Petersb. med. ztschr., 1871, n f., ii, 306, 350.

LANE: The Kinks which Develop in Our Drainage System in Chronic Intestinal Stasis, Brit. Med. Jour., 1911, i, 913.

LANGER: Lehrbuch der Anatomis des Menschen, Wien, Braumüller, 1865.

LANZ: Der McBurney'sche Punkt, Centralbl. f. Chir., 1908, xxxv, 185.

LEICHTENSTERN: Verengerungen; Verschliessungen und Lageveränderungen des darms, In; Ziemseen's Handbuch., Leipzig, Vogel, vii, p. 359.

LIEPMANN: Eine Bauchfellduplikatur zwischen dem Mesosigmoideum und dem weiblichen Geschlechtsapparat; das "Ligamentum infundibulo-colicum, Virchows Arch. f. path. Anat., 1912, ccvii, 352.

LITTLE: Internal Strangulation; Anatomy of the Vermiform Appendix, Dublin Jour. Med. Sc., 1871, lii, 237.

LOCKWOOD AND ROLLESTON: On the Fossæ Round the Caecum, Jour. Anat. and Physiol., 1892, xxvi, 130.

LONGYEAR: Nephrocoloptosis, St. Louis, C. V. Mosby Co., 1910.

LUSCHKA: Die Anatomie des Menschen, ii, Der Bauch, Tübingen, Laupp, 1863, p. 139.

Ueber die peritoneale Anheilung des Blindarmed und über die Fossa iliocæcalis, Virchows Arch. f. path. Anat. u. Physiol., 1861, xxi, 285.

MALL: Development of the Human Intestine and Its Position in the Adult, Bull. Johns Hopkins Hosp., 1898, xx, 197; Transl.: Arch. f. Anat. u. Entwcklngsgesch., 1897, Suppl. vol. ccc, 403.

MARSHALL AND EDWARDS: Agenesis of the Vermiform Appendix, Philippine Jour. Sc., 1906, 1, 1061.

MAXIMOW: Experimentelle Untersuchungen über die entzündliche Neubildung von Bindegewebe, Fischer, Jena, 1902.

MCBURNEY: Experience with Early Operative Interference in Cases of Disease of the Vermiform Appendix, New York Med. Jour., 1889, l, 676.

MERKEL: Topographische Anatomie, Braunschweig, Viemeg. ii, 565.

MONKS: Intestinal Localization, Ann. Surg., 1903, xxxviii, 574.

MONRO: Medical Essays and Observations, Edinburgh, Ruddimans, 1733-44, iv, Art. ii, p. 57.

MOYNIHAN: On Retroperitoneal Hernias, Balliere, London, Tindall & Cox, 1899.

NAGEL: Handbuch der Anatomie des menschen, v. Bardeleben, Fischer, Jena, vii, 2nd Part.

NAGEL: Harn und Geschlechtsorgane, Jena, Fischer, 1896.

NORRIS: The Omentum; Its Anatomy, Histology and Physiology in Health and Disease, Univ. Penn. Med. Bull., 1908, xxi, 119.

RAMSTROM: Anatomische und Experimentelle Untersuchungen über die lamellösen Nervenendkörperchen im peritoneum parietale des Menschen. Anat. Hefte, 1908, xxxvi, 309.

RANVIER: Traite technique d'Histologie, Savy, Paris, 1889.

REID: Studies in the Intestine and Peritoneum in the Human Foetus, Jour. Anat. and Physiol., 1911, xlv, 73.

RETZIUS: On the Semilunar Lines of Douglas, Edinburgh Med. Jour., 1858, iii, 865.

ROBINSON: The Peritoneum, W. T. Keener & Co., Chicago, 1900.

ROCHER: Disposition anormale de l'insertion du mésentère, Jour. de méd. de Bordeaux, 1904, xxxiv, 45.

SAMSON: Einiges über den Darm, insbesondere die Flexura sigmoidea, Arch. f. klin. Chir., 1892, xliv, 146, 386.

SANDIFORT: Tabulæ intestini duodenali Lugd. Bat., 1780, p. 7.

SANTORINI: Tabulæ suptum decem, Santorini, Parma, 1775.

SAPPEY: Description et Iconagraphie des Raisseaux Lymphatiques, Paris, 1886. Des vaisséux lymphatiques, Traité d'anatomie, Paris, Delahaye, 1869, ii, 760.

SAVAGE: The Surgery, Surgical Pathology and Surgical Anatomy of the Female Pelvic Organs, ed. 3, New York, Wm. Wood & Co., 1880.

SCHIEFFERDECKER: Ueber einen Fall von rudimentärem grossen Netz beim Menschen und über die Bedeutung des Netzes, Deutsch. med. Wchnschr., 1906, xxxii, 988.

SCHOTT: Beiträge zur Anatomie der Fossa iliocœcalis, Wchnbl. d k. k. Geselch d. Aertz. in Wien, 1862, xviii, 345.

SCHRIDDE: Ueber den angeborenen Mangel des Processus Vermiformis, Virchows Arch. f. path. Anat., 1904, clxxvii, 150.

SÉRNOFF: Zur Kenntniss der Lage und Form des mesenterialen Teiles des Dünndarmes und seines Gekröses, Internat. Monatschr. f. Anat. u. Physiol., 1894, xi, 437.

SNOW: Case of Strangulation of the Ileum in an Aperture of the Mesentery, London, Med. Gaz., 1846, n. s., iii, 1049.

STOPNITZKI: Zur Untersuchungen Anatomie des menschlichen Darmes, Internat. Monatschr. f. Anat. u. Physiol., 1898, xv, 219, 327.

SUZUKI: Ueber die Resorption im Omentum majus des Menschen, Virchows Arch. f. path. Anat., 1910, ccii, 238.

TARENETZKY: Beiträge zur Anatomie des Darmkanals, Mém. Acad. de St. Petersburg, 1881, Série vii, 28.

TIMOFEJEW: Ueber die Nervenendigung in Bauchfell und in dem Diaphragma der Säugeltieren, Arch. f. mikr. Anat., 1902, lix, 629.
TREITZ: Hernia Retroperitonealis, Prag. F. A. Credner, 1857.
TREVES: The Anatomy of the Intestinal Canal and Peritoneum in Man, London, Lewis, 1885; Hunterian Lecture also in Brit. Med. Jour., 1885, i, 415, 470, 527, 580.
TUFFIER: Étude sur le cæcum et ses Hernies, Arch. gén. de méd., 1887, i, 641; ibid, ii, 52.
TWYMAN: Nonrotation of the Ileum with Fatal Intestinal Obstruction Due to Occlusion of the Ileum by the Overriding Colon, Jour. Am. Med. Assn., 1918, lxx, 672.
VALLIN: Situation et prolapsus des ovaires, Thése de Paris, 1887, No. 266.
VIRCHOW: Historisches, Kritisches und Positives zur Lehre Unterleibsaffectionen, Virchows Arch. f. path. Anat., 1852, v, 281.
WALDEYER: Das Becken, Bonn, Cohen, 1899, p. 300.
Eierstock und Ei, Leipzig, Engelmann, 1879.
Die Kolon-Nischen, die Arteria colicæ, und die Arterienfelder der Bauchhöhle, Berlin, G. Reimer, 1900.
WALDEYER: Hernia retroperitonealis nebst Bemerkungen zur Anatomie des Peritoneums, Breslau, Junkfer, 1868.
WEGNER: Chirurgische Bemerkungen ueber die Peritonealhöhle mit besonderer Berücksichtigung der Ovariotomie, Arch. f. klin. Chir., 1877, xx, 64.
WEINBERG: Topographie der Mesenterien und der Windungen des Jejunoileum beim neugeborenen Menschen, Internat. Monatschr. f. Anat. u. Physiol., 1896, xiii, 66, 89.

CHAPTER V

WOUND HEALING (THE FORMATION OF FIBROUS TISSUE)

The integrity or strength of any organ is dependent on its fibrous tissue. It is the solution of continuity of this tissue that constitutes the essential lesion when an organ is injured. It is the restitution of the continuity of fibrous tissue that constitutes the primary and most important factor in the repair of wounds. In wounds of the skin for instance epithelial tissue can not regenerate itself unless preceded by the healing of fibrous tissue. In the injury of the highly developed epithelial tissue, as for instance the kidneys, liver or brain, the process of healing is confined to restoration of continuity of the connective tissue stroma.

In harmony with this general principle the peritoneum, when injured, repairs itself chiefly through a growth of its fibrous tissue. The integrity of the abdominal cavity is dependent on the peritoneum and the fibrous tissue of the gut wall. It follows then, that, whenever the surgeon violates this integrity or it becomes severed by disease, he must produce mechanical conditions which will artificially secure this end temporarily and place the tissue in a favorable state for permanent repair, before he has secured his patient against certain disaster. The principles involved in securing this result, therefore, form the foundation of abdominal surgery. It is the purpose of this chapter to trace the processes of healing as they take place in the injured peritoneum. It is only by understanding the various phases of this that we can hope to avoid the things that are inimical to its rapid fulfillment.

The theory of the process of connective-tissue formation as here set forth is in some respects wholly new. It is based on the study of the process of the healing of wounds as it is observed to take place in the abdominal cavities of mammalia. While it can not claim to be complete as a biological study, it explains the repair

of injury as it takes place in this tissue with a detail sufficient for the use of the practicing surgeon.

The advantage of employing the organs within the abdominal cavity for such study lies in the fact that here we deal with an exclusively mesenchymal tissue in a region where it is possible to exclude bacterial infection. This enables us to study separately the process of repair from the reaction of inflammation. In the studies in wound healing heretofore recorded, this has not been attained.

Our literature still bears the strong impress of the period when the healing of infected wounds alone was studied. Since that day an understanding of the influence bacteria play in the healing of wounds has become the universal property of the surgical mind, but pathologists have not seen fit to extend the study of the processes of repair as pertains to the bacteria-free, as contrasted with the bacterially contaminated, wound. Even surgeons have been short-sighted in that they have focused their concern wholly on bacteria and the harmful influence they exert in the healing of wounds. The subtler influences of chemical factors, other than those produced by the growth of bacteria, have not yet received adequate recognition, largely perhaps because it has as yet eluded complete analysis. While we can not as yet identify these chemical factors, their influence must be recognized as vitally modifying the healing process. These influences do not excite reactive processes, as bacteria or physical agents do; nevertheless, their baneful influences must be recognized.

When the processes of inflammation and wound-healing coexist the latter can become active only after the former has overcome the noxious agents. The tissues possess a certain capacity to prevent the development of bacteria, but the conflict in the tissues may be so slight that it does not find expression in marked subjective or objective changes generally recognized as inflammation. The surgeon is apt to depend too much on the ability of the peritoneum to take care of a certain amount of infection because animal experimentation and clinical experience have shown that it is capable of doing so. Did he realize that by just so much is final healing retarded, he might manifest a greater reluctance in

adding the additional burden. Devitalized tissue is as important as bacteria in producing minor disturbances in wound healing.

It is of interest to observe the differences between ideal and slightly disturbed wound-healing. This can be done in a very exact way in the series of experiments about to be detailed. It is possible here to observe these lesser influences which delay but do not prevent healing. These factors are important in operations on the gut tract because delay here may permit serious complications to develop.

Ideal healing can take place only when there are no foreign substances, in the form of either bacteria or dead tissue, to disturb the process. The development of fibrous tissue in an aseptic environment has, heretofore, been the exclusive right of the embryologist. His material, naturally, has been quite different from that which concerns the surgeon. In the embryo the tissue develops slowly and in continuity, instead of quickly over a limited extent, as occurs in the repair of a wound.

Between the study of the origin of fibrous tissue as dealt with by the embryologists, and the study of healing of infected wounds studied by the pathologist, lies naturally the entire lifetime of the patient, and the time of the surgeon's activities lies somewhere between. The study of the fibrous-tissue formation, as it takes place in aseptic wounds in fully developed tissue naturally falls to the lot of the surgeon because he alone has a vital interest and he alone comprehends the technic which makes it possible to secure such wounds.

It can not be too strongly emphasized that pathologists have not correlated the modern conception of the relation of wound-healing and the coincident reaction to the experiences of the surgeon. The cause of this has been twofold: in the first place, the early processes of healing and bacterial reaction, being both responses to injury, are exceedingly closely related; and pathologists have not recognized how widely divergent the lines become, starting as they do at so small an angle. Surgeons, viewing the process from a greater distance, have long known that wound-healing and bacterial inflammation, as observed in the peritoneum particularly, are the antithesis of each other. The whole fabric of abdominal surgical practice is based on the empirical demonstration of

the fact that this is true. It is the purpose of this and the subsequent chapters to discuss the scientific basis for this fact.

Theories of the Development of Connective Tissue.—Before proceeding with a presentation of my own opinions on the formation of fibrous tissue in aseptic wounds it is worth while to examine the views held by embryologists and pathologists, and to note, briefly, the avenues by which they arrive at the conclusions they have asked surgeons to accept.

Two theories of the origin of connective tissue are generally recognized. One group of observers contends that the fibers form within the protoplasm of the cell, and are later extruded from it. There is a difference of opinion as to whether or not this takes place throughout the protoplasmic mass or only in the peripheral zone of the cell. The other group of observers contends that the fibers form in the intercellular substance from substances produced by the cell. To these may be added my own theory which may be briefly stated as follows: Fibrin fibrils form in an amorphous exudate, these fibrin fibrils are transformed into connective-tissue fibrils.

At the head of the group who believe that the fibrils are formed within the protoplasm of the cell unquestionably stands Schwann. He noted in the study of pig embryos of 4 to 7 cm. in length, that the cells elongate at opposite poles, and break up into tufts of fine fibrillæ. He believed that this process continues until the whole cell has been changed into fibrillæ. Valentine modified this theory by assuming that each fiber is the product of a bipolar prolongation of a spindle-form cell. It may be said for this theory that in the later stages of fibrous tissue-formation in infected wounds pictures may be observed which tend to confirm it.

This theory was supported by the studies of Virchow and Donders and later by Schultze.

Lwoff modified Schwann's views inasmuch as he believed that it was only the surface of the cell that was concerned in the development of fibrillæ. This investigator tried to answer one objection to the cell-protoplasm theory, namely, that individual cells acting independently could not produce long fibers, by assuming that a number of cells, acting in conjunction, produced a single fiber. He denied that the cell protoplasm breaks up into fibrils.

Spuler concluded that the fibrillæ first form within the cell, and later become extruded by the cell retracting from them, thus confirming fully the work of Schwann.

Boll is quoted generally as favoring the cell protoplasm origin of the fibers; but a reading of his article shows clearly that in some situations (developing cartilage) he notes especially that no connection with the cells can be traced.

Flemming, while generally adhering to the view that the fibrillæ are formed from the surface of the cell, is undecided as to their relation to the long fiber bundles between the cells. He states that these bundles probably are formed by the fibrins from the cells being united by some chemical change, but it is possible that they may be formed from the intercellular substance. Mall summarizes the work of the earlier investigators, and agrees with those who believe that the fibrils are formed from the periphery of the cell. He employed both frozen sections and digestion methods in order to isolate the fibrils.

Henle was the first to contend that the connective-tissue fibrillæ were derived from the ground substance. Kölliker held that the fibrillar elements develop in the formless material between the cells. The cells, according to him, played a metabolic role, but did not take part in the formation of the fibers.

Merkel is the most recent contender for this theory as developed in the study of embryonal material. Studying the process in the umbilical cord, he noted that the fibers developed without any contact with the cells. He holds that the course of all connective tissue is the cell syncytium of the mesenchyme. This is composed of a colloidal substance which fills the spaces in the cell network. Wherever this comes in contact with other tissues it becomes condensed into an amorphous layer. These layers are without cells, but cell processes may extend into them. In these layers the fibrils form. In the beginning the fibers are not collagenous, being still granular.

The conclusions reached from a study of embryonal material are dependent upon the personal equation of the observer. The facts may be summed up as follows: Early, the future connective tissue is made up largely of cells. The intercellular substance later rapidly increases. Within the homogeneous intercellular substance

fibrils or prefibrils appear. These prefibrils are attached to the cells or the cells to them, as the observer may wish to state it. These fibrils may be traced into the cell protoplasm, and they may extend long distances into the intercellular protoplasm, reaching other fibers or surfaces. The source of this intercellular substance is generally assumed to be the cell, though no one has offered proof of this. That fibrils are attached to some cells appears probable, but, on the other hand, fibrils which can not be traced to any cell exist in abundance in many situations. Though anatomists will have nothing to do with my theory of the formation of fibrous tissue, I fail to see a great difference in point of fact between their findings and mine. The conclusions reached differ more widely than the recorded observations. This seems true particularly of Merkel's work, published some years after my own. Embryologists, it seems to me, suffer a bias from a too long association with pathologists and their infected material. It would perhaps be more correct to state that the early embryologists were themselves pathologists, and furnished their own bias. Be this as it may, the changes observed in embryonal tissues do not parallel those observed in wound healing, since the latter takes place much more rapidly; and surgeon-pathologists are at fault in trying to harmonize their findings with those of embryologists.

Tissue development comparable to that seen in the embryo may be observed in hemorrhages into organizing tissue. I have studied it particularly in hemorrhages into myomas. Here pictures are obtained which are exactly parallel with those observed in embryonal tissue, as pictured by numerous observers (notably Mall, see Figs. 5 and 12 in reference). The source of the "intercellular substance" in the myoma is unquestionably the blood stream. The changes in the blood vessels which precede the hemorrhage in these tumors are of a hyaloid nature. Because of the similarity in the histochemistry of the tissue in the embryo and the myoma, I venture to suggest to embryologists that the intercellular substance at the period when fibrous tissue is being more abundantly formed is not necessarily a product of the cells, but that, possibly it may be derived from the blood stream.

Pathologists have reached conclusions parallel with those of the histologists just quoted. Marchand expresses himself as follows:

"It seems to me certain that the formation of the intercellular substance takes place only in immediate contact with cells, and is derived from direct transformation of the particles of the cell-bodies themselves." He makes this statement after admitting the possibility of the origin of connective-tissue fibrils from the intercellular substance. Hamilton by the use of sponge grafts noted that fibrin bundles formed between the tissue and the foreign body. He observed that the cells made their way along these bundles. The significance of these observations made by Hamilton has been generally overlooked. Wounds in which grafting is undertaken are relatively free from active infection; therefore, they approach in a measure the processes as seen in aseptic wounds. His description of the relation of the migration of cells to the formation of fibrin bundles forms a very worthy preliminary to the *in vitro* observations made by Burrows.

Ziegler came fully to substantiate the conclusions of Schwann, Schultze and Boll. Stricker believed that the fibers were developed from cells, but that cells under the influence of inflammatory reaction could revert to an embryonal state. From this substance, then, fibers may form.

Aufrecht noted the sparseness of connective-tissue cells in some fields in wound healing. Most pathologists, however, who have studied wound healing have studied the cells and noted the fibrils extending from one or more poles of the fibroblasts and from these preparations made their deductions.

The Healing of Aseptic Wounds (Healing by First Intention).—The broader aspect of wound healing will first be considered under the abstract consideration of the formation of fibrous tissue. By proceeding in this manner the problem is simplified; and, so far as practical considerations go, this covers the essential factor, for the salutary and baneful factors which act on wound healing are dependent entirely on the effects they may have on the development of the fibrous tissue. The cellular activity, interesting from a theoretical standpoint, might be wholly disregarded without suffering loss.

Though little employed in the past, obviously no more favorable tissue could be obtained for the study of the genesis of fibrous tissue than in aseptic wound healing, for here the process runs its

course much more quickly than in either embryonal or inflamed tissues. Among wounds those of the peritoneum offer a particularly inviting field for such study since all of the processes go on in a moist chamber, as it were, subject to our inspection at any period we may wish to observe it.

A further advantage of a study of this tissue lies in the fact that the process is rapid. The solution of any physical-chemical process in nature often depends on finding a way to exaggerate one or other of its phases to make them stand out prominently. In the embryo the formation of fibrous tissue is a relatively slow process, while in the wound it not only takes place independently of the formation of the other elements, but also at a rapid rate.

I have no quarrel with the views held by embryologists. This study was undertaken wholly from a surgical standpoint. I sought to determine how adhesions form and why some are permanent while some loosen after a certain time. It was the result obtained in this connection that has justified this more general discussion of the nature and development of fibrous tissue. The immediate practical results as applied to surgical practice lead me to believe that former views have been built on erroneous concepts of the cell and its functional significance. Recent general knowledge of life processes and the newer methods of study have given us a better insight into the whole nature of the process.

The following experiments were planned to permit the study of fibrous tissue under the most favorable conditions. The material employed was obtained by uniting peritoneal surfaces by suture, either by intestinal anastomosis or by simply suturing two peritoneal surfaces together. This was supplemented by the study of omental adhesions formed by suture or by a mechanical irritation produced by placing foreign bodies against a surface of peritoneum and securing them there by means of sutures. Peritoneal membranes which formed between opposed surfaces or over these foreign bodies furnished a most instructive material for the study of the development of connective tissue. Corn pith, discs of cork, and cakes of paraffin were the substances most used.

There is no dividing line between the formation of fibrous tissues, the healing of wounds, and the formation of adhesions. Practical consideration makes it desirable that the adhesion-formation

be considered in a separate chapter. For an understanding of the fundamental laws governing both they must be considered together.

The studies upon which the conclusions were made cover a wide range. Most of them were made, naturally, by animal experimentation; but in the course of surgical practice much material was obtained which served greatly to supplement the experimental studies. These observations served chiefly to demonstrate that animal experiments warranted conclusions which can be carried to the human subject with but little modification.

Having determined that minor modifications were dependent on the age and species of the animal, error was reduced to a minimum by employing litters of animals. As a matter of routine six animals of the same litter were experimented on. Different series were employed to study the different periods. For instance, one series was observed at 1, 2, 4, 8, 16, 32, hours and another at 1, 2, 4, 6, 16, and 32 days. Such series were employed to determine the fundamental factors. Other series varying from 10 minutes to 40 days were observed in order to determine the extreme limits. The technic employed in all intraabdominal manipulations was that common to surgical practice.

Supplemental to the above, organization of blood-clots also was studied. Preparations were obtained by tying off a segment of a vein full of blood. After ligation, clotting and organization follow. Such preparations were studied at intervals varying from 2 hours to 4 weeks. These experimental studies were supplemented by the study of the organization of clots in human vessels. Some of these were of uncertain data while the life cycle of others could be determined with the accuracy of an experiment, as surgeons will readily understand.

In order to eliminate as nearly as possible the personal equation from these observations, the experiments were repeated each year for ten consecutive years before the theory here advanced was published as a preliminary report in 1904. The results then published have been confirmed by frequent repetitions of the studies since that time.

The Formation of Fibrin Bundles.—The fundamental factors in the formation of fibrous tissue may be observed in the simplest

Fig. 97.—Exudate in the angle between two peritoneal surfaces twenty-four hours after suture. Oc. 2, Obj. 2/3.

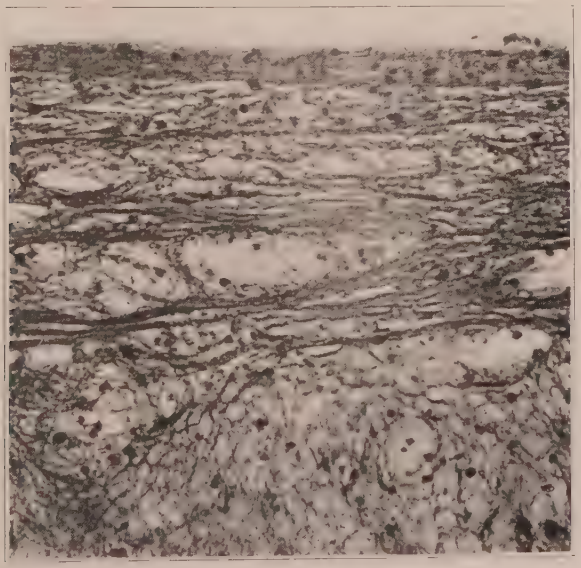

Fig. 98.—Parallel arrangement in the primitive fibrin bundles twenty-four hours after anastomosis.

form by examining a section across the line of suture after an intestinal anastomosis has been made according to the Czerny-Lembert method. In such preparations it may be observed that,

as soon as the peritoneal surfaces are brought into contact by the sutures, an exudate forms, filling in the angles between the two apposed planes of the peritoneum (Fig. 97). On the surface of this exudate a layer of fibrin is formed. This layer of fibrin forms in bundles arranged parallel to each other (Fig. 98). Below the surface other bundles may form either parallel to the first or in a network beneath (Fig. 99). I have observed the formation of

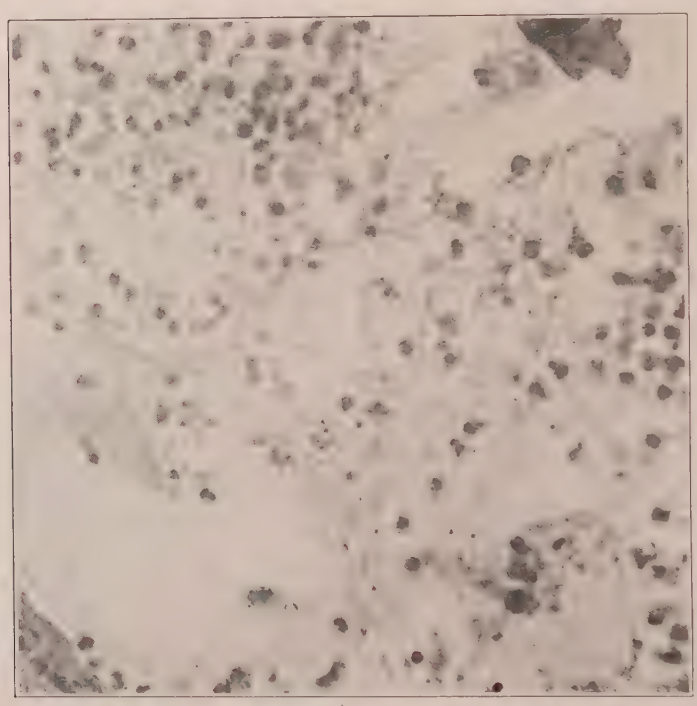

Fig. 99.—Network of fibrin formed below the parallel fibers as in Fig. 97.

a similar network in the interior of hemorrhages in myomata. Burrows explains the formation of tension points. Baitsell recently confirmed this opinion by the study of clotting as observed by aid of dark-field illumination. The significance of this will be discussed later. These bundles may be demonstrated as early as ten minutes after the suture has been applied, though none are fully formed until thirty minutes have elapsed. The process in the uncomplicated cases is usually complete in about two hours.

If irritation is continued, new fibrin bundles within certain limits may be produced.

These bundles extend from one surface to the other, not in a straight line but in a gradual curve, not unlike the cables in a suspension bridge or the trabeculæ of the neck of the femur; in other words, they represent stress lines. This arrangement is uniform for exposed surfaces, no matter where the suture is placed. This law applies as well to wounds in solid viscera and to those of the skin.

These fibrin bundles may be produced in an even more striking

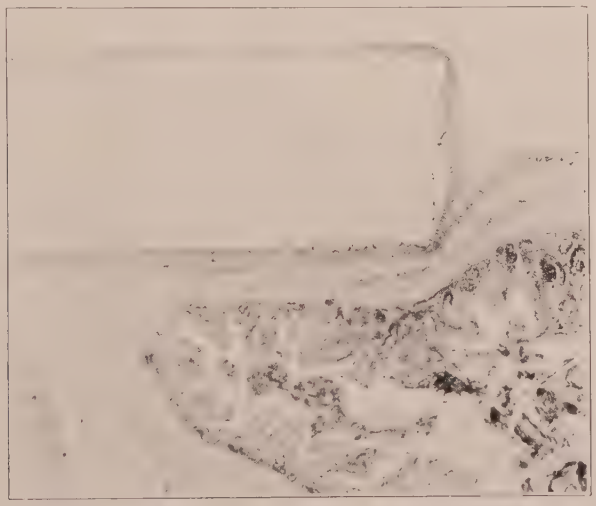

Fig. 100.—Corn pith attached to a loop of gut. Parallel bundles have formed over its entire surface.

way, as follows: When a disc of corn pith or other foreign body is attached to a surface of peritoneum the entire object is soon covered by an amorphous exudate (Fig. 100). This exudate quickly covers the entire surface of the disc in a uniform layer. Fibrin bundles develop over the entire surface of the foreign body simultaneously. These fibrils also assume the direction of an engineer's cable.

The rapidity with which this exudate forms is one of the most surprising observations in this experiment. It may be studied by

sectioning the specimen after sacrificing the animal soon after the experiment is performed, or by observing the process through a glass window. It can readily be observed in performing anastomosis in the human subject. In doing a gastroenterostomy, for instance, the first line of sutures will be surrounded by this exudate by the time the operation is completed. The same thing may be observed whenever a peritoneal wound has been caused, as after insults by the forceps.

The bundles spanning the angles between opposed surfaces have been referred to as fibrin bundles. The assumption that they are such is justified by both histochemical and physiologic studies. The histochemistry was studied by means of the various staining methods recognized by histologists. Nearly all the recognized stains that react specifically to fibrin were used. After trying various methods, Weigert's fibrin stain, Mallory's connective-tissue stain and Van Gieson's picrofuchsin were employed on each tissue studied. Weigert's stain is perhaps the most nearly specific, but is the most difficult to employ. Mallory's stain is useful in the study of end-results, but uncertain in the study of the transitional stages noted below. When one becomes accustomed to the picrofuchsin method it is the most satisfactory for general use, for in the gradually changing shades of color the process of transformation can be followed accurately. Some of the details of this study may be briefly presented. The early bundles stain with Weigert's method (Fig. 101). This stain does not act in specimens older than forty-eight hours. At this early period the fibers stain a brownish red with Mallory's stain. At from the third to the fifth day the heavy bundles shown (Fig. 98) begin to break up into fiber fibrils. They begin at this time to take the blue dye of Mallory's stain (Fig. 102). At this time they do not yet take the red of Van Gieson's stain. The interesting point is that this change occurs simultaneously over any surface. For instance if a piece of corn pith one cm. in diameter is stitched to the peritoneum the fibers covering the center of this disc will undergo these changes simultaneously with those at the border (Fig. 103). None of these fibers take the fuchsin of Van Gieson's stain before about the seventh to the tenth day. So far as tinctorial study permits one to judge, the bundles are

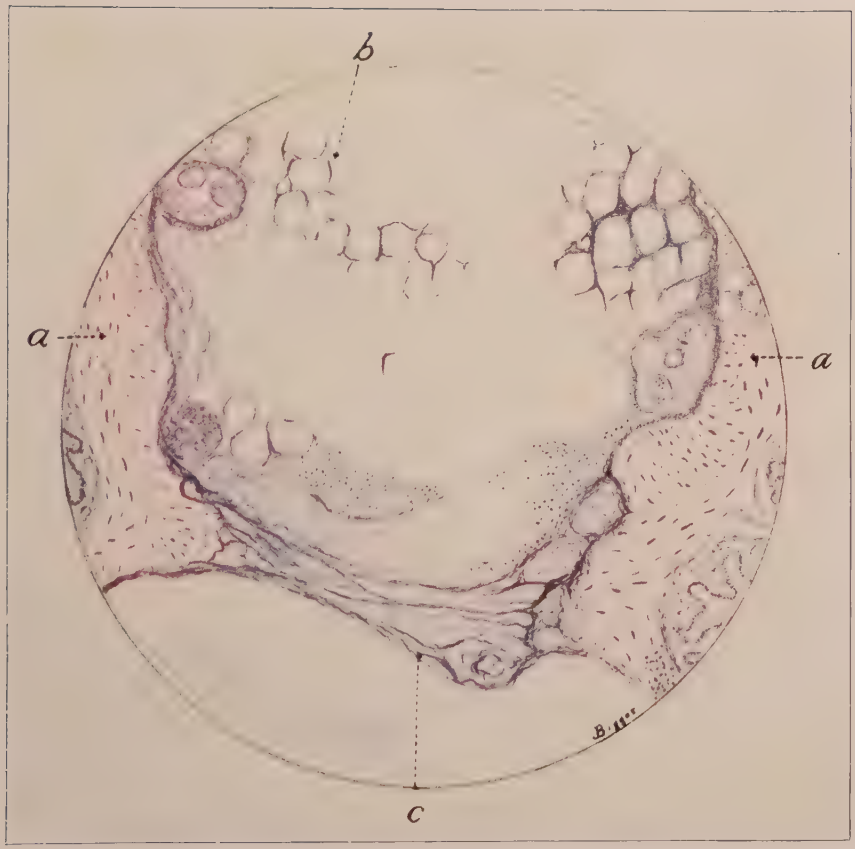

Fig. 101.—Bridge of fibrin formed over the edge of a bit of corn pith lying between two gut surfaces. Weigert's stain, twenty-four hour specimen. *a*, wall of the intestine; *b*, corn pith; *c*, fibrin bundles.

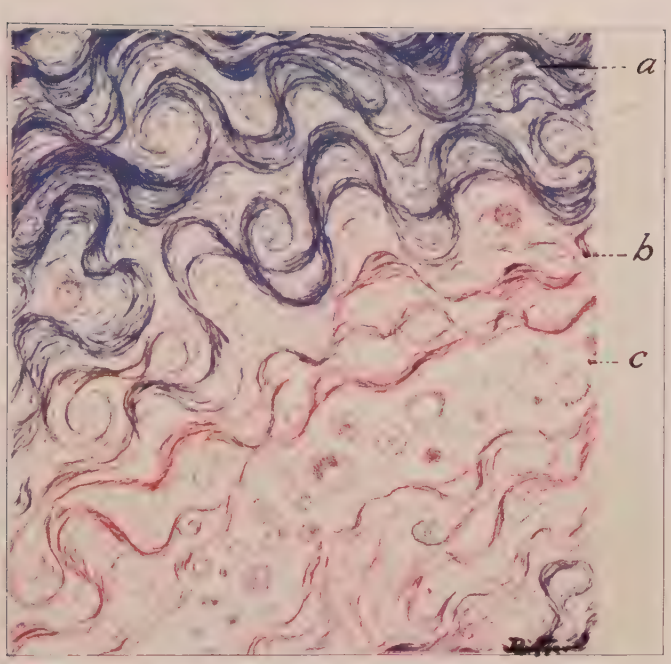

Fig. 102.—Fiber bundles extending between two peritoneal surfaces. Mallory's stain. Six-day specimen. Oc. 2, Obj. 1/12. *a*, fibers formed staining blue; *b*, fibers in process of development still staining reddish brown; *c*, newly formed blood vessel.

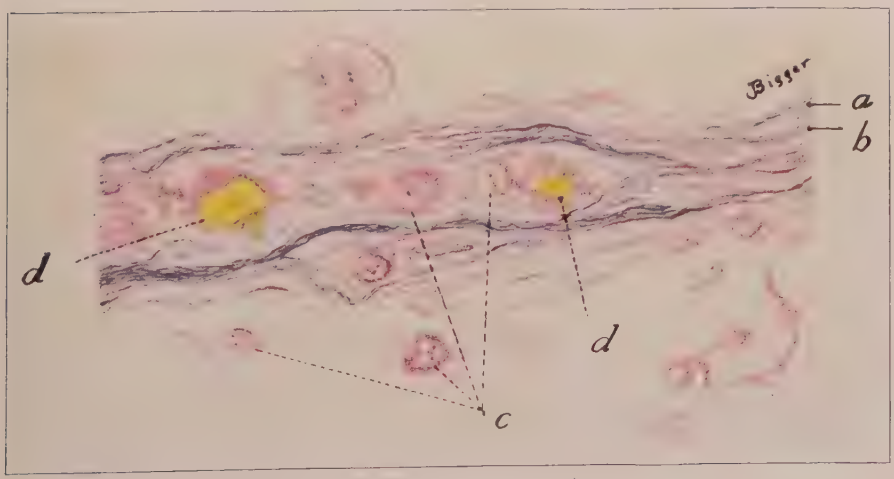

Fig. 103.—Fibrils removed from the center of a disc of corn pith. Six-day specimen. Oc. 2, Obj. 1/12. *a*, fibers staining reddish brown; *b*, fibers staining blue with Mallory's stain; *d,d*, foreign bodies.

fibrin. They stain yellowish with Van Gieson's, reddish-brown with Mallory's and blue with Weigert's fibrin stain.

In order to check up the accuracy of the tinctorial reaction physiologic methods that are known to prevent the coagulation of blood, that is, prevent the formation of fibrin, were employed. Leech extract and peptone were used most extensively. Magnesium sulphate was used to some extent, but was found to be too toxic. As an example of the method of employing this method the following

Fig. 104.—A disc of cork placed against the parietal peritoneum is covered with a new peritoneum after four weeks.

may be cited; if 0.3 gm. of peptone per kilo weight of the animal is injected intravenously or intraperitoneally, and an anastomosis is then made, the process of healing, as above described, does not take place. The exudate forms, but the fibrin bundles do not develop. Likewise, if the corn discs are soaked in peptone solution before being placed in animals otherwise untreated, these bundles do not form. It is worthy of note that the inhibitory influence of the peptone is only temporary, and is soon, usually in from four to nine

hours, overcome by the animal, after which the fibrin bundles form. If sections are made before this time either an amorphous exudate or a granular fibrin will be found. The peptone acts as a repellant to leucocytes, as well as prevents the formation of fibrin. The process by which the inhibiting power of the peptone is overcome is not known. Fresh pancreatin extract acts in a similar manner,

Fig. 105.—Magnified wedge of Fig. 104 showing the complete blood supply.

and a crushing wound of the pancreas, made at the time of the operation, may act in the same way.

Both by tinctorial study, therefore, and by aid of methods which are known to control the formation of fibrin, the results were uniformly positive for fibrin. It seems warranted in assuming therefore, that the bundles that formed as above indicated are fibrin.

The extent of the new tissue formation was one of the surprises

of this study. A cork disc a centimeter and a half in diameter is covered by fibrin within a few hours. The entire surface is covered simultaneously. A membrane of any extent is completely formed within a few weeks (Fig. 104). The extent and the completeness of the vascularization is shown in Fig. 105. The same complete vascularization takes place when the disc is attached by one pole only (Fig. 106). These membranes when removed show the same structure as a normal peritoneum.

The Fate of Fibrin Bundles.—This much demonstrated or assumed, as the reader may prefer to believe, the question that re-

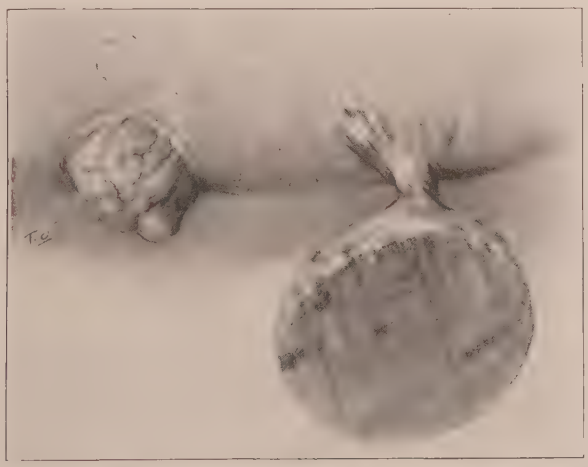

Fig. 106.—A cork disc attached to one point in its circumference. The entire disc is covered with a new membrane. At the left of the figure is a cotton pledget also covered with a new membrane.

mains is to determine what becomes of the fibrin bundles. This problem was studied by taking series of young rabbits, as already noted, and studying the tissues removed at varying intervals and making tinctorial studies. The changes observed are as follows: the fibrin bundles give the typical reaction of fibrin to all specific dyes up to twenty-four hours. After this time they no longer stain typically by Weigert's stain, but still take Van Gieson's and Mallory's stain typically. At three to five days strands among the bundles begin to take the blue of Mallory, while the remainder stain brown. As time progresses, up to seven or eight days, the

number of fibers staining blue by Mallory increases until all so stain. The typical red stain by the Van Gieson method is not obtained for some days after they stain blue by the Mallory method. With Mallory's stain the fibers either take the blue completely or not at all. It is particularly interesting to observe the bundles of which one segment stains brown and the remainder blue, with this stain. With Van Gieson it is different, in that the change is gradual, the color shading off into various degrees of intensity as it approaches the red. When one once becomes familiar with the Van Gieson stain it is more valuable in studying the various stages of fiber development than either of the other stains above mentioned.

The relation of the connective-tissue fibrils in the healed wound to the preliminary fibrin bundles must remain problematical. The only way in which this relation can be determined, aside from the tinctorial studies above noted, is by studying the relations of these bundles to the cells in a large series of experiments.

The first striking factor is that the final fiber bundles invariably retain the same topographic relation to the peritoneum and the foreign body which the fibrin bundles assumed. If the fibrin bundles are replaced by fiber bundles, the latter are identical in arrangement with the former. The assumption that the fibrin bundles are taken up by the so-called fibroblasts and that the fibers are laid in their place by these cells, can readily be answered by sections in which the change of fibrin to fibrils is taking place in the absence of cells. This can be seen over the surface of segments of corn pith and often in organizing blood clots (Fig. 107). In these situations often the tissue change may be seen to go on with exceedingly few cells in the entire field. In the small cavities, in corn pith, for instance, one may find fibrils undergoing the tinctorial changes when no changes are present. Most conclusive of all, perhaps, are those specimens in which long bundles are observed which stain half red and half blue with Mallory's method (Fig. 103). These facts led me to believe that the change from fibrin to fibrils may take place without the actual physical contact of cells, though no doubt what chemical influence their presence in the neighborhood exerts is another question. I am not prepared from my material to go quite as far in denying the influence of cells as Baitsell goes. There seems to be a limit to which fibrils may

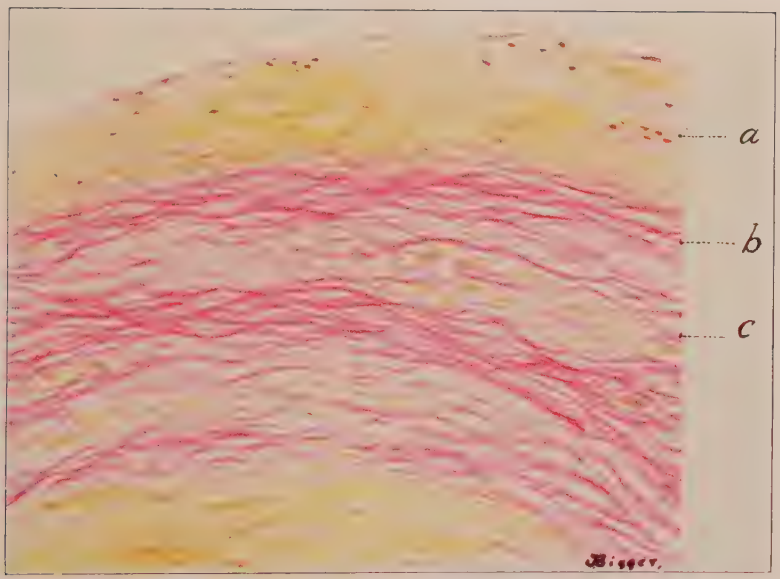

Fig. 107.—Organizing blood clot. Connective-tissue fibers have formed with the presence of relatively few cells. Human material. Two weeks' duration. Van Gieson stain. Oc. 2, Obj. 1/6. *a*, fibrin bundles staining yellow; *b*, connective tissue fibrils staining red; *c*, small spindle-form "fibroblasts."

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form in the absence of cells, or, at least, to the extent one can find cell-free areas.

Notwithstanding the fact that the studies stopped short of a positive demonstration, the result of these studies caused me to formulate the theory that the initial factor in wound healing is the formation of fibrin bundles, and that these bundles remain permanently, and, by virtue of certain changes, become fibrous tissue. It is only on this basis that the rapid healing of wounds as observed in the peritoneum, can be accounted for. It answers also the conditions as observed in skin grafting. This conception makes it clear why the use of any drug or any solution applied to any wound acts to retard healing.*

My own belief in the correctness of the theory above presented is based most of all on the fact that it explains the phenomenon observed in the conduct of wounds under clinical conditions. Scientific workers, as a rule, have a contempt for such evidence, but it is a matter of medical history that, unless a theory conforms to the facts as presented by the clinical course of the disease, it is not enduring. The theory here announced is in conformity with the facts as noted in actual conditions of practice, the healing of wounds, the failure of wounds to heal, and the formation and the permanency of adhesions.

In recent years evidence which tends to confirm the theory above advanced has appeared. During 1908 M. T. Burrows was a coworkers in my laboratory and had the opportunity of observing the series of experiments I conducted during the summer. Later Burrows undertook the culture of tissue, and I spent some time with him while he was an associate of the Rockefeller Institute. The results arrived at by him seemed confirmatory, and I used the information thus gained in a paper published in 1913. He had noted that the fibrin fibrils, after a considerable growth of connective tissue, stain the characteristic color of the fibers of white fibrous tissue with Van Gieson stain. Baitsell also has made a study of the transformation of fibrin into fibers in the living culture, and seems to confirm the findings of Burrows. He makes

*This observation, it may be noted here, is in harmony with what is noted in the subperitoneal hyperplastic inflammations, as seen particularly about the cecum. The presence of fibrin remaining in an unchanged state seems sometimes to be the starting point of sarcoma in hemorrhagic myomata.

the additional observation that the newly formed fibrous tissue reacts like embryonic connective tissue when treated with pancreatin, and differs from adult connective tissue in that the latter is not digested by pancreatin. My observations along this line have shown that the susceptibility to pancreatin is definitely present during the first ten days (in the rabbit), but that after a time (thirty days), it is as resistant to pancreatin as is adult fibrous tissue. Furthermore, the resistance to pancreatin is parallel in a general way with the affinity for acid fuchsin. If fibrous tissue takes this stain it will not be acted on by pancreatin. Comparing my results with Baitsell's I am disposed to assume that the complete change does not take place in cultures or that this author did not follow the cultures a sufficient length of time, or, as he himself suggests, that the tissue experimented upon reacts differently from human tissue. Save for this final step these studies fully confirm my own. His words will bear quotation: "The study of prepared sections shows that at first, in the coagulation tissue formed in the wound as a result of the clotting of blood and lymph, a typical fibrin net is present. Later this fibrin net is transformed into fibrous tissue containing bundles of wavy fibers, in which, in many instances, the individual clot becomes transformed into this new fibrous tissue. This transformation of the clot and the formation of the fibrous tissue is not due to any intracellular action, inasmuch as it takes place before the connective-tissue cells wander into the coagulation tissue. It is a direct transformation of the fibrin clot, and is identical with the process which was previously found to take place in the fibrin clots in living cultures of adult frog tissues and which have been noted above. The connective-tissue cells which wander into the newly formed fibrous tissue from the cut edges do not digest the fibers, but move among them and evidently cause a breaking up of the larger bundles of fibers into smaller ones, and later we have formed, as a result, a tissue which is typical in appearance with regular connective tissue. The connective-tissue cells are, in many cases, rounded cells when they first appear in the fibrous tissue, but later they assume the typical elongated-spindle shape of fibroblast cells. This change in shape appears to be due to the action of the cells in stretching along the fibers. The preparations do not show any

connection between those spindle-shaped cells and the fibers which are already formed, nor is there any evidence of any attempt by them to form new fibers intracellularly."

Burrows has extended these studies along other lines, and, since he has not yet published all of his studies, I avail myself of the permission to quote fully from his notes, supplemented by my memory of the work as I have followed it through the years we have conducted independent but related studies. I do this because these studies cast a hopeful light on the abnormal, as well as on the normal, development of tissue and I do it particularly because they emphasize the part the cell plays in certain phases of tissue development. This is important because the more the formation of fibrous tissue departs from the normal, the more important becomes the part the cells play. This range includes the formation of fibrous tissue, on the one hand, and the development of sarcoma, on the other.

Burrows undertook to solve this problem of the cell by a direct analysis, considering the formation of connective tissue as a part of a general physicochemical reaction in which the growth of the cell is a factor. He drew his conclusions from theoretical physicochemical reasoning. The adoption of this method for solution was made on the assumption that the formation of fibrous tissue is a reaction different from cellular growth because in the scar the cells lie only against the fibers that are wholly extracellular.

Burrows determined that the curve of the growth of connective-tissue plasma cultures, stated in terms of amount of growth and time, is that of the law of mass action. In a tissue culture the growth commences after a given latent period. It proceeds rapidly for a time, and later gradually ceases. In the wound the reaction proceeds in identically the same way, although the cells do not degenerate, but simply come to rest. In a limited number of cultures he found that the cells do the same. He found that this condition of inactivity with disintegration of the cells took place only in those cultures where the cells had wandered directly into the clot and had become completely surrounded by pure fibrin, separated as a dense mass of fibrils from the serum portions of the clot.

Cells which migrate along the surface of the clot often grow very actively. These are not in direct contact with fibrin. Their growth curve is the same as the other except that after growth ceases they invariably degenerate. The cells within the clot and surrounded by fibrin, he found he could keep in the incubator in the presence of oxygen for six months, which corresponds again to what is observed in myomata. Cells go on to full development when the fibrin changes to fibrils. It is possible that in the stimulus of the change in the fibers on the cell to development lies the reason that cells in healing wounds do not go on to malignancy.

In the early studies on tissue cultures Burrows noted that these cultures were not comparable to cultures of bacteria. Bacteria grow at the expense of the medium. No medium capable of imparting life to body cells has been found. He came to this conclusion because the growth was not proportional to the medium about the cells, but to the size of the fragment, provided the fragment was not larger than 1 mm. in diameter, the thickness being limited by the depth to which diffusion readily could take place. Oxygen, for instance, diffuses readily and in sufficient quantity to maintain metabolism more than 0.5 mm. into compact tissue or fresh blood clot.

Single cells do not grow. In 1911 Carrel and Burrows transplanted fragments from one medium to another to find that they grew only through the few transplants. Burrows measured the growth of each transplant to find that it was never as great as the original mass of tissue transplanted. The growth observed was a simple transfer of material from the cells disintegrating in the center of the fragment to those on the periphery. The number of new cells formed on the periphery amounted to the original mass minus the energy transfer.

It seems to me that some of the cells in the tissue cultures, which are regarded as newly formed cells, are but those residing in the tissues, and escape from the fragment into the surrounding plasma. Burrows has determined that individual cells when isolated do not multiply, yet the demonstration of this very fact is highly desirable before we can accept in full the conclusions of tissue growth *in vitro*. I fail to find any evidence that cells multiply *in loco* in inflammatory lesions, as in healing wounds. I

believe all the cells concerned in these processes are manufactured in central arsenals, and are transported to the field of action. This explanation is not satisfactory even to myself, I hasten to remark, but I enter it here merely to draw forth more exact expressions from those engaged in the culture of tissue.

The Relation of Cells to the Formation of Fibrous Tissue.—Unquestionably, the important factors in wound healing are chemical in nature, but the intricacies of the process transcend existent knowledge. Any statement relative to the part the cells play in the process as above detailed must be based largely on studies of morphology. Such studies heretofore published seem to me to reflect only the author's personal equation. I have repeated many of the studies of recent years, notably those of Maximow, Schridde, and Weidenreich, but have failed to establish to my own satisfaction a single fact that could be interpreted in terms of wound healing. In my opinion the large endothelioid cells are identical with the macrophages and likely with Maximow's polyblasts. At any rate, he says of these cells, that they "may be round, polymorphous, large or small, may stain dark or light, may show granulations in their bodies, are very numerous in inflamed young connective tissue, in the formation of which they play an enormous role." He does not clearly differentiate between this group and plasma cells. In this respect he strikes a sympathetic cord in me, for the same cell appears different when the environment changes. For instance, in edematous inflamed tissue the plasma cells take on much fluid and their nuclei stain less deeply, making their differentiation from endothelial cells difficult. Ranvier's clasmatocytes, I feel sure, are endothelial cells.

Mallory disposed of a group of cells having resemblance to mononuclear leucocytes, on the one hand, and endothelium, on the other, by calling them endothelial leucocytes. In this way he indicates the close relation of endothelial cells and mononuclear leucocytes. It would not be too much to call them transition forms. More recent studies by anatomists, notably those of Stockard, give play to a wide latitude of speculation in the cytology of wound healing. My own studies along this line have not passed beyond the stage of personal enjoyment, and are not worthy of record here.

Notwithstanding the unsatisfactory results of my own studies

on the cell content in wound healing, I venture to append some fragmentary observations, because no others have been recorded which deal with the kind of tissue used in my experiments. The number and kinds of cells present in healing tissue depends much upon the degree of reaction the tissues undergo in the early stages of the process. If much hemorrhage attends the manipulations, or if there is much crushing of tissue, and particularly if there is infection, the number and variety of cells is much enhanced.

The condition in its simplest form, without the hemorrhage or infection and with the minimum of trauma, will first be considered. With the first escape of plastic material a few cells are always encountered. Theoretically, the process is possible without the advent of cells, as I have noted in wounds of the capsule of the liver, but practically the technic in the peritoneum is too difficult to be executed without producing some degree of reaction. The cells that are observed in the fibrin bundles are mononuclear leucocytes for the most part, but a few polynuclears are always present. These cells arrange themselves in columns along the bundles of fibrin or in the meshes of the secondary fibrin network.

That this is a sequential process and that the fibrin bundles are there first and the cells glide along them, was confirmed by Burrows in his tissue culture experiments. I had previously noted that a complete column of large mononuclear cells forms on the surface of the mass continuous with the surface of the peritoneum. These are globular at first, and become gradually flattened out as the underlying fibrin undergoes its specific changes.

Burrows found that the connective-tissue cell, leucocytes and epithelial cells growing in cultures rounded off when placed in liquid portions of the medium. But if connective-tissue cells were placed against a surface where cells were growing, they flattened out as oil droplets flatten on the surface of water. It was only the cells in contact with surfaces that flattened out, while those lying in the fibrin meshes became elongated or spindle-shaped and those lying within the fibrin meshwork became stellate, their processes extending along the various threads of fibrin. Conversely, if an elongated spindle-shaped cell was transferred to a flat surface it became flattened spindle- or polyhedral-shaped.

These studies are in harmony with what is observed in the heal-

ing of wounds in the peritoneum. Early in the process the cells covering the surface and those lying more deeply are identical. The subsequent fate of these cells seems to be purely a matter of location (Fig. 107). Those on the surface become ultimately endothelial cells, while those lying more deeply elongate and become typical connective-tissue cells. It is interesting to note that the change in cell-form is always parallel with the change of tinctorial reaction of the fibrils and corresponds to the stage of the healing process. If the changes in the fibers are delayed the change in the cells also is delayed. The question which factor, the cell change or fiber change, has the power of initiative is still problematic, though I feel confident the latter is the potent factor.

The cause of the changes in the cells is problematic. Burrows believes that it is a question of the availability of oxygen. The physical factors governing the growth of cells, he believes, is the same, but that different cells differ in the amount of oxygen required for their development. He believes that this oxidation reaction is a simple chemical reaction, and, as the reaction must be incomplete and that it ceases when the product of reaction reaches a certain concentration, just as fermentative reactions cease when the amount of alcohol produced reaches a certain concentration.

He found that cell activity would be renewed by carefully transplanting them to new media. Since the renewing the CO_2 with a stream of air had no effect on the curve, Burrows was led to believe that the stoppage of growth was due to the accumulation of waste products of the metabolism of these cells, the body cells being like many other living cells in that they produce more than one substance in their process of oxidation.

His previous experiments were performed largely by the use of the tissue of chicken embryos cultivated in the plasma of chickens. The blood of birds is peculiar in that it does not clot readily when removed from the body free from any contamination with tissue. He found that drops of this plasma, which remained fluid even in the incubator, clotted quickly after the addition of a tissue fragment. In this primary clotting it did not change in volume, but changed merely from a fluid-like state to a structureless jelly. Such preliminary clotting could be brought about by adding any

tissue fragment, even after heating to 60° or even a 100° C. for several minutes. Such clots underwent no further change unless a fragment containing living cells and oxygen was added. In this case after a latent period, varying with the age of the tissue of from two to twenty-eight hours, they commenced to contract. The contraction took place first about the fragment and involved only a small part of the clot near it. This led him to believe that primary clotting and clot contraction were different phenomena, and that they were dependent on different conditions.

These studies are interesting in that they furnish some explanation as to the reason some sterile wounds do not heal. Surgeons know that an abdominal incision, for instance, may heal without infection, without perceptible reaction of any sort, yet the resulting scar may be weak, become stretched, and a hernia form. From Burrows' studies we may assume that such failure in the healing process is due to the accumulation of waste products in the area of the wound, which have the power of inhibiting the healing process. Whether this inhibition affects alone or primarily the cell, or whether it acts by preventing chemical interchanges in the fibrin bundles, is not cleared up by these experiments. My own studies permit me to say that in such instances there is a failure of the fibrin bundles to undergo the normal change. Why they do not do so can not be determined by morphologic studies.

That healing is inhibited is demonstrated by the fact that when such slumbering cells are transplanted to a new medium they begin a renewed growth.

The physical relation of the cells to the fibrin bundles has been discussed. What their chemical relation is can not be stated with like confidence. That these cells give off their protoplasmic substance toward the actual formation of the fibers, as the old theories would have us believe, is unlikely, for the changes in the fibers above detailed take place for large areas in which there are no cells. In corn pith one can note the transformation of fibrin into fibrils in compartments where there are no cells, as already stated. Grönroos noted that in the large meshes of the embryo of the cat there are no cells, and no indication that they have any part in the development or in the maintenance of the nutrition. Baitsell also has noted this. That cells have been there and then migrated

is hardly possible, for had they taken part in the development of the fibers, as the theories would have us believe, migration subsequently would hardly have been possible.

The cells that gain access to the fibrin clefts in specimens in which the conditions for healing are ideal, are for the most part cells which contain oblong nuclei often with two prominent nuclei. The protoplasm is usually abundant, and the cells lying in the meshes may present processes varying in number and degree in the later stages, but early they are more or less spheroidal. These are undoubtedly endothelial or at least endothelioid cells. What relation these cells bear to the large mononuclears of the blood stream I am unable to say. My guess is that the endothelioid cells of the fixed tissues and in wound healing are these mononuclears grown up. In the later stages the cell with the processes appear. They correspond to the classical fibroblasts. It is only in infected tissues, however, that the typical fibroblasts appear in numbers. Whether the cells seen in granulation tissue are endothelioid which have given off their protoplasm and become spindleform or whether they are of other genesis, is difficult to say.

According to our old teaching it would be altogether reasonable to assume that this sacrifice of cellular protoplasm has to do with the formation of fibrin. Whether or not we are to assume a continuance of this influence is another problem. Even in the areas in which the change takes place in the absence of cells it is quite possible that the cell elaborates substances which are carried by processes of diffusion to the area in which the changes are taking place. These statements are suggested as a compromise with the views of those who adhere to the old theories. The final solution must await a more extended knowledge of chemistry.

Burrows has noted that a preliminary clot undergoes secondary contraction in the presence of connective-tissue cells, but that, when oxygen is replaced by hydrogen or nitrogen, or when heated to 60°, the contraction ceases. I have already made record of the fact that the use of hot packs is inimical to the most salutary healing of wounds. It would seem, therefore, that the healing process is dependent on some living factor, and is not a purely chemical one. The fact that a temperature 60° C. represents about the borderline of resistance of agglutinins reminds one that there may

be a relation of this process to that of certain properties of sera. I feel, therefore, that one should hesitate before going to the full extent that Baitsell goes in denying *any* influence on the part of the cell in the process of the change from fibrin to fibrils.

In just what way cells contribute to the transfer of fibers from fibrin is not yet clear. Burrows believes the food-materials are derived from the cells and not from the medium. If this is true the cells must be able to transmit their fiber-producing substance to the medium, because in some situations where fibrinogenesis is going on rapidly the cells are not numerous enough to act directly, but may serve as an activating substance, on the surrounding fluid medium.

Be that as it may, all these studies are in agreement that the primary factor in wound healing is the formation of fibrin bundles, and that these bundles are transformed directly into fibrous tissue by agencies as yet unknown. These are the facts for which I have so long been contending.

Regeneration of the Endothelium.—The fate of the cells which cover the surface of the fibrin bundles can be determined with much confidence. The process of endothelial regeneration has been studied on cross section and also by silvering and then studying them on the flat. Upon the outermost layer of fibrin a layer of large mononuclear cells arranges itself (Fig. 108). Sometimes this layer is several cells deep (Fig. 109). When this is the case all cells disappear save a single layer. These cells retain their large nuclei with their prominent nucleoli, and there is no loss of cell protoplasm. These form the endothelial cells of the final healed surface (Fig. 110). The cells within the meshes of the fibrin, identical at first with those just mentioned, undergo extensive changes. At first spheroidal, they gradually flatten, and become progressively more spindle form. As the healing process becomes more advanced the covering endothelium gradually reaches the final flat stage, while those cells lying deeper become progressively more spindle form (Fig. 111).

That the endothelium of the surrounding surface of the peritoneum has any direct part in the covering of these surfaces can not be demonstrated. Furthermore, the entire surface becomes en-

dothelialized simultaneously, and not gradually, from the border, as in the epidermitization of skin wounds. This precludes the possibility of a regeneration from the border. For instance, a

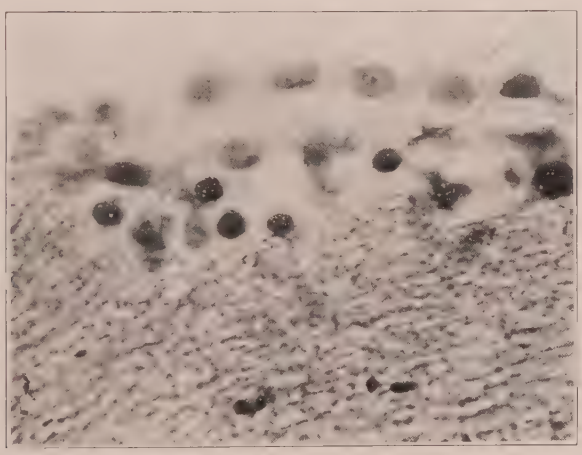

Fig. 108.—Large mononuclear cells about fibrin bundles in a superficial wound of the peritoneum. Oc. 2, Obj. 1/12.

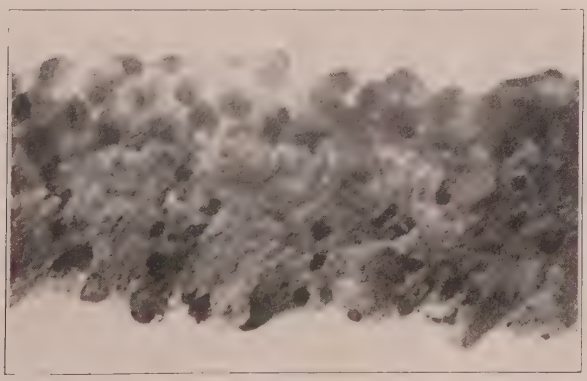

Fig. 109.—Several layers of large mononuclear cells covering a layer of fibrin. Oc. 2, Obj. 1/12.

cork disc, 1 cm. across, receives its endothelial covering at its center at the same time that its edges are so covered.

Effect of Trauma on Fibrous Tissue Regeneration.—When traumatism is inflicted on the area undergoing the healing process the

number and variety of cells throughout the healing process is much increased. In the milder degrees the number of cells is but slightly increased and the amount of exudate is unchanged. In extreme degrees of trauma granular fibrin alone is formed, the polynuclears predominate, and the fibrin fibers do not form at all. In such instances healing does not begin until secondary reactive processes have prepared the way. In that event the interference

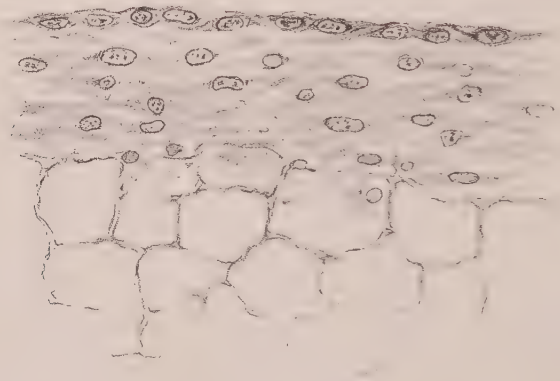

Fig. 110.—Endothelium of a newly formed peritoneum in the process of reaching the final stage. Oc. 2, Obj. 1/12.

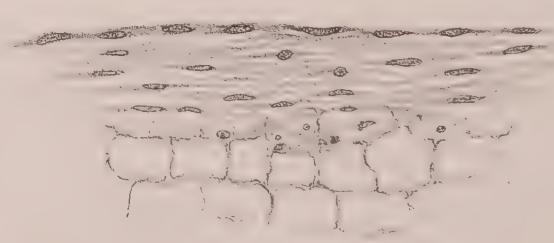

Fig. 111.—Cells in their final resting stage. Oc. 2, Obj. 1/12.

may be as great as in the case of bacterial infection. This is particularly true when there has been an escape of many red corpuscles, for these appear to delay the formation of fibrin nearly as effectually as does the presence of an active infection. The mere puncturing of a vessel does not produce those results. It seems to require, in addition, a liberation of a ferment from the crushing of fibrous tissue. The end result of this condition differs markedly from infection in one point; namely, the irritation is not progressive as in

the case of bacterial infection. These observations are of importance for they furnish a scientific basis for the generally recognized principles that all tissues should be handled gently if we expect expeditious healing of wounds.

Healing of Infected Wounds (Healing by Second Intention).— The preceding account of the formation of fibrous tissue was essentially a discussion of wound healing by first intention. The existence of an aseptic state of the wound is the prime essential. Not only is freedom from bacterial contamination a prerequisite, but absence of any physical or chemical factors inimical to the initial formation of fibrin is presupposed. Not all wounds are free from such contamination. In event of such contamination another series of phenomena takes place. In this class of wounds fibrin formation takes place imperfectly or not at all. When this is true we speak of healing by secondary intention. While this nomenclature is an adaptation from the older literature it is particularly apropos to this study, for here it signifies that the fibrin bundles preliminary to the formation of fibrils must take place as a secondary process. It happens, therefore, that the classification, adopted long before infection even was understood, fits perfectly the knowledge of the present day.

Modern study of wound healing dates from the studies of John Hunter, who was the first to recognize the coagulation of lymph as an important factor in the process. It is this substance that made healing by primary intention possible. When this was not possible, according to him, inflammation must ensue in order to provide the material for healing. If this does not take place, then suppuration, with subsequent formation of granulations must ensue. We have, therefore, his division of the healing of wounds in three stages: First, healing by the coagulation of blood between the edges of the wound; second, healing by inflammation; and third, healing by suppuration. The second and third are subdivisions of the healing by secondary intention, as defined by Hippocrates.

Hunter's classification shows an acuteness of observation not equaled by any other observer. It emphasizes a point not generally recognized, namely, that a failure to heal by first intention may occur, and yet an actual suppuration may not follow. This inflam-

matory nonsuppurative class represents a degree of disturbance in which the primary formation of fibrous bundles is prevented, with a consequent institution of the reactive processes, which stop short of actual suppuration. That is to say, it is not active enough to produce a degree of destruction visible to the naked eye, because the infection is overcome by the defensive forces of the body before it reaches this degree. The processes which may bring about this intermediate stage are vast in number. The use of hot water in a wound will bring it about, as will any of the so-called antiseptic solutions. Any agent in fact that brings an irritating foreign body between the edges of the wound may act in this manner. It is not generally enough recognized that the destruction of tissue by rough operating leaves foreign bodies in the form of dead protoplasm, which causes a reaction in kind, if not in degree, as that produced by bacteria.

It is equally important to understand that slight degrees of disturbance of healing may take place as a result of bacterial growth not great enough to produce suppuration. The disturbance may be too slight to manifest itself by a general disturbance or even a local change, but it may cause a slight induration or a slight reddening about the wound. This induration indicates a conflict in which serous exudate and leucocytic infiltration takes place. An exudate of this character does not permit the production of a fibrillar fibrin, but a granular one. When such a state exists the irritant substance must be removed before the healing process can proceed. The delay in healing caused by these inconspicuous disturbances may be greater than when actual suppuration occurs. Incoagulable exudate of any character may prevent healing for days, weeks or even months.

A part of the wound may heal ideally, while another part may show disturbance. Tissue with a great capacity for reaction may remove the noxious factors in a short time, while an adjacent tissue less favorably equipped may fail to heal for a much longer time. This difference can be illustrated by the healing of an abdominal incision. The peritoneum and fascia through the subperitoneal vessels being well supplied with vessels, readily overcome a slight irritation, and soon heal. The skin also may soon recover and unite. The muscle, and particularly the adipose layers, being less well

fortified, are unable to remove the noxious elements, and healing may be delayed. This delay may permit the growth of feeble bacteria; and the familiar late subacute abscess results. It follows that these feeble tissues must receive our special care if we are to avoid trouble of this kind.

These principles apply with especial force in the healing of peritoneal surfaces. The peritoneum is so provided with circulation that it is the most efficient of all tissues in controlling noxious agencies. In this respect the different regions exhibit a varying capacity. A peritoneal surface abundantly underlaid with fat is less efficient than a like region not so incumbered. Regions where the peritoneum is closely attached to fascial planes have less regenerative and protective capacity than one lying on a fibrous tissue substratum in which vessels can freely course.

So many of its injuries involving the peritoneum permit the escape of intestinal contents. This implies contamination of the wounded surfaces, not only with a great variety of bacteria, but with the fermentative juices of the gut tract. Any injury as from fault in technic on the part of the operator may so cripple the defensive forces that the balance of power in the contest against the agencies above noted may be disturbed, resulting in delayed healing, with all that implies. These disturbances may not find expression in the subjective feelings of the patient nor on the clinical chart, and yet may be pregnant with the greatest significance for the future well being of the patient.

Gross disturbances with pus formation are well understood. When the edges of a wound are the site of active suppuration, whether the result of perforating disease or from faulty technic, the formation of granulations is the necessary outcome. These processes are well understood by all who operate. This granular tissue may be observed in any wound that has harbored a drain for a week. These exhibit the triumph of defensive factors over infection.

Though the history of healing by secondary intention has been written from surface wounds it is both fitting and important in this connection to study them as they occur in the abdominal cavity. It is here, too, where healing by inflammation, as contrasted to healing by suppuration, can best be studied.

Healing by Inflammation.—Under this head I mean to include

those disturbances in wound healing which stop short of suppuration. On superficial inspection these wounds may seem to run an undisturbed course but when examined in detail they show many deviations from ideal wound healing. The wide difference, for instance, between a perfectly executed anastomosis and an imperfect one often can not be appreciated without a microscopic section of the healing wound. The causes of the lesser disturbances in wound healing as observed in the peritoneal cavity are many. These may be separated into those groups which manifest a different type of reaction. Though these divisions are in no wise to be considered as distinctly separate, they are sufficiently so to permit recognition by the surgeon and they can be produced experimentally with a certain degree of regularity. The various types may be mentioned in the order of clinical occurrence.

Irritation from Infection.—In this group are included all those conditions where healing is delayed because of the presence of microbes or their products. The type is most frequently observed when anastomoses are made. Bacteria commonly reach the surfaces that are apposed by the suture after the lumen is opened. Or they may reach the surface through a stitch canal after the suture has been completed, bacteria may gain access to the fresh wound by entering through the imperfectly apposed mucosa.

The result of such infections may be one of three degrees of disturbance. In the lesser degree there is an abundant round-celled infiltration in the deeper portions of the wound while the preliminary fibrin bands on the surface of the wound remain undisturbed (Fig. 97). The infected area is composed of granular fibrin, many leucocytes, serous exudate, and bacteria. When the infection occurs later fibrin formation takes place normally but is destroyed by the toxins before complete development can be reached. The result is that the peritoneal surface forms a fibrous covering and the deeper tissues gradually cicatrize as the infection is conquered. This type really represents a combined primary and secondary healing. The superficial portion heals by primary intention, while the deeper heals by secondary. This is the course that most anastomosis wounds pursue.

When a greater infection occurs the entire exudate between apposed surfaces shows the effect of the infection (Fig. 98). No-

where are the fibrin fibers completely formed. It is only when renewed exudation takes place after the infection has been subdued, that healing takes place. This period can be roughly said to take from three to ten days. During this period the sutures prevent, in large measure, a separation of the surfaces. It is in this type of disturbed healing that permanent sutures exert a saving influence. When absorbable material has been used, the wound edges may separate, with the attendant disaster.

The third degree is represented when infection of the entire wound occurs and an absorbable suture is used, or when nonabsorbable sutures have been used and the infection is relatively greater, and the period of suppuration exceeds the period during which the permanent suture holds the apposed surfaces in coaptation. The result is that the sutured edges separate and the infection extends entirely through the suture line and invades the surrounding peritoneal cavity. The patient is sometimes saved by omental or other organs becoming adherent to the suture line. The infection when so walled in is retained within limits until the healing process by granulation can be inaugurated, and carried to completion.

This type is characterized by infiltration of the tissue about the suture line. The subperitoneal tissue is increased many times in thickness, the increase in volume being due to a serous exudate and a great abundance of polynuclear leucocytes. Bacteria can always be demonstrated in these areas. Later, when the period of secondary fibrin formation has taken place, large mononuclears are present in large numbers.

Irritation by Ferments.—When healing is delayed by the escape into the wound of the digestive ferments there is no perfect formation of fibrin. There remains then a fluid exudate poor in any cellular elements and deficient in fibrous elements. This is very apt to extend throughout the coapted surfaces at the point of the anastomosis. In this type of irritation no attempt at healing takes place until the ferments are overcome by secondary exudation from the peritoneal surfaces. If all agglutination is prevented bacteria may escape along the passage so left, and may cause an invasion of the general peritoneal cavity unless prevented by secondary adhesions of other organs.

This type of disturbance is most apt to occur in the upper ab-

domen where the intestinal ferments are most active. The same disturbances may occur when ferments reach the line of suture, as when the pancreas is wounded.

The most striking changes occur when this type is associated with bacterial infection, as is sure to occur if healing is long delayed. In this type there is an enormous thickening of all the tissues involved in the wound. It is not uncommon to find the stomach wall a centimeter thick, and the small gut nearly as much. The infiltration is made up of serum and many large round cells with abundance of protoplasm, resembling much the signet-ring cells observed in Krukenberg's tumors of the ovaries. In its physical characters this infiltration reminds one of a woody phlegmon, or the chronic indurations not infrequently observed in wounds after operations for hernias. This thickened condition may exist for many months, and, if great enough, may imperil the fecal circulation of the gut.

Irritation from Necrosis.—Under this heading may be included all destruction of tissue the result of technical accident such as injudicious bite with the forceps, or a too tight ligature, or a puncture of a vessel resulting in its subsequent occlusion by thrombus with the attendant necrosis of the area so nutrified. In this type the disturbance usually affects only a small segment of the suture line. These accidents are most apt to occur at the mesenteric border in end-to-end anastomoses or at the end of the wound in lateral anastomoses.

In this type when the necrotic tissue is small it is effectually walled in by the healing wound; when large enough to include the entire thickness of the gut wall perforation may occur at the time the dead tissue separates from the living. This disaster may be anticipated by previous adhesions of adjoining organs to the threatened spot.

In each of these types there is an absence of suppuration; and the loss of substance, except sometimes in the last-named group, is only microscopic. There is always a reaction to injury, followed by a renewed attempt at wound healing. This is where reaction and healing touch together separated only by the narrowest of lines.

Healing by Suppuration.—This type of healing by secondary intention is preëminently that of disease processes. When a perfora-

tion occurs, infection of the environment ensues and suppuration is inevitable. There is usually a loss of substance which must be filled in. Here is where the three tasks of healing recognized by Marchand, namely, the defect in the fibrous tissue must be bridged, parenchymatous tissue may have been destroyed and must be substituted for, and, finally, an exudate on the serous surface may have formed and if unobservable, must be converted into fibrous tissue.

The difference between healing by suppuration and healing by granulation is only one of degree. We speak of suppuration only when that degree of reaction is reached where exudate, leucocytes, and tissue debris reach an amount that it is perceptible to the naked eye, in other words, when pus is produced.

When infection is too great for fibrin bundles to form, the healing process must proceed by the formation of granulations. This is brought about when the leucocytes so far gain the ascendency over the infection that fibrin can be formed between them and the unaffected tissue. Along these fibers mononuclears creep, to be followed by the formation of new vessels. These, in turn, supply new serum, and healing is advanced by a repetition of the process. When the degree of sterility of the wound is reached that the formation of fibrin on the surface of the wound is no longer prevented, union between the granulations of the apposed surfaces takes place.

In this way an opening, for instance, in the appendix, is closed, and the continuity of the gut is restored.

If the muscle wall of the gut is destroyed the bulk of the defect, since muscle is capable of but feeble regeneration, must be made up by the formation of new connective tissue. If this area is great, owing to the tendency of young connective tissue to contract, serious constriction of the gut's lumen may be caused.

Sometimes an exudate on the surface of the peritoneum undergoes such changes that absorption is not possible. These insoluble products excite an exudate, which becomes fibrous. This exudate may accumulate within the layers of the peritoneum or a mesentery. In either situation contraction follows with constriction of the underlying bowel.

Healing by suppuration may take place about a sutured gut when the degree of infection outside the bowel is set up, which

results in the formation of a pus cavity. It is not unusual for this to occur in perforation of the gut, or in a suture line when the degree of infection is great. This abscess may break into the gut from which the infection comes, as it usually does, or it may break into the free peritoneal cavity with attendant disaster. The subsequent course of such a process is discussed in the chapter on adhesions.

Bibliography

Aufrecht: Ueber die Genese des Bindegewebes, etc., Virchows Arch. f. path. Anat., 1868, xliv, 180.

Baitsell: A Study of the Clotting of the Plasma of Frogs' Blood and the Transformation of the Clot into a Fibrous Tissue, Am. Jour. Physiol., 1917, xliv, 109.

The Origin and Structure of a Fibrous Tissue Which Appears in Living Cultures of Adult Frog Tissues, Jour. Exper. Med., 1915, xxi, 455.

The Origin and Structure of a Fibrous Tissue Formed in Wound Healing, Jour. Exper. Med., 1916, xxiii, 739.

Boll: Untersuchungen über den Bau und die Entwickelung der Gewebe, Arch. f. mikr. Anat., 1872, viii, 28.

Burrows: Some Factors Regulating Growth, Anat. Rec., 1916-17, xi, 335.

A Method of Furnishing a Continuous Supply of New Medium to a Tissue Culture in Vitro, Anat. Rec., 1912, vi, 141.

Grafting of Normal Tissues as Dependent on Individual Affinity: Autoplastic, Isoplastic, Heteroplastic, Tr. Internat. Cong. Med., 1913, Sect. iii, Gen. Path. and Path. Anat., 217.

Wound Healing in Vitro, Proc. New York Path. Soc., 1913-14, xiii, 131.

The Tissue Culture as a Physiological Method, Tr. Cong. Am. Phys. and Surg., 1913, p. 77.

Cultivation of Human Cells in Vitro, Med. Rec., 1914, lxxxvi, 649.

An Attempted Analysis of Growth, Anat. Rec., 1915, ix, 64.

The Oxygen Pressure Necessary for Tissue Activity, Am. Jour. Physiol., 1917, xliii, 13.

Some Factors Regulating Growth (Abstr.), Anat. Rec., 1916-17, xi, 335.

Carrel and Burrows: Cultivation of Tissue in Vitro and Its Technic, Jour. Exper. Med., 1911, xiii, 387.

Donders: Form, Mischung und Function der elementären Gewebetheile im zusammenhang mit ihre Genese, Ztschr. f. wissensch. Zool., 1851, iii, 348.

Flemming: Zur Entwicklungsgeschichte der Bindegewebsfibrillen, Internat. Beitr. z. wissensch. Med. Festschr. R., Virchow, 1891, i, 213.

Grönroos: Bindegewebe ohne Bindegewebeszellen, Anat. Hefte, 1903, lxviii, 137.

Hamilton: On Sponge Grafting, Edinburgh Med. Jour., 1881, xxvii, 384.

Henle: Allg. Anat., 1841, p. 197-379.

Hertzler: Peritoneal Adhesions, Their Cause and Prevention, Tr. West. Surg. Assn., 1904, xiv, 76.

The Principles of the Technic of Skin-Grafting, Interstate Med. Jour., 1913, xx, 135.

Pseudoperitoneum, Varicosity of the Peritoneum and Sclerosis of the Mesentery, with a Preliminary Note on the Development of Fibrous Tissues, Jour. Am. Med. Assn., 1910, liv, 351.

The Development of Fibrous Tissues in Peritoneal Adhesions, Anat. Rec., 1915, ix, 83.

KÖLLIKER: Würzburg med. Verhandl., 1853, iii, 1.

Die embryonalen Keimblatter und die Gewebe, Ztschr. f. wissensch. Zool., 1884, xxxiv.

Die Bedeutung der Zellenkerne für die Vorgäuge der Vererbung, Würzburg, 1885, Reprint from Ztschr. f. wissensch. Zool., xlii.

Bernerkungen zu E. Hackels Aufsatz über Ursprung und Entwicklung der thierischen Gewebe Sitzungsb. d. phys.-med. Gesellsch. zu Würzburg, 1885, p. 49.

Ueber den feineren Ban des Knockengewebes, Sitzungsb. d. phys.-med. Gesellsch. zu Würzburg, 1886, p. 33.

Ueber die Nichtexistenz eines embryonalen Bindegewebskeims (Parablasts), Sitzungsb. d. phys.-med. Gesellsch. zu Würzburg, 1884, p. 14.

LWOFF: Ueber die Entwicklung der Fibrillen des Bindegewebes w., Sitzungs. d. k. Akad. d. Wissensch. Math, Natur, 1889, xcviii, 184.

MALL: On the Development of the Connective Tissues from the Connective Tissue Syncytium, Am. Jour. Anat., 1901-2, i, 329.

MALLORY: The Principles of Pathologic Histology, W. B. Saunders Co., Philadelphia, 1914.

MARCHAND: Der Process der Wundheilung, Enke, Stuttgart, 1901.

MAXIMOW: Experimentelle Untersuchungen über die entzündliche Neubildungen vom Bindegewebe, Fischer, Jena, 1902.

Weiteres über Entstehung, Structur und beränderungen des Narbengewebes, Beitr. z. path., Anat. u. z. allg. Path., 1903, xxxiv, 153.

MERKEL: Betrachtungen über die Entwicklung des Bindegewebes, Anat. Hefte, 1909, xxxviii, 321.

RANVIER: Des clasmatocytes, Compt. rend. Acad. d. sc., 1890, cx, 165.

SCHRIDDE: Die eitrigen Entzündungen des Eileiters, Fischer, Jena, 1910.

SCHULTZE: Ueber Muskelkörperchen und was man eine Zelle zu nennen habe, Arch. f. Anat. u. Physiol., 1861, p. 1.

SCHWANN: Mikroskopische Untersuchungen über die Uebereinstimmung in der Struktur und dem Wachsthum der Thiere und Pflanzen, 1839, p. 137.

SPULER: Beiträge zur Histologie und Histogenese der Binde- und Stutzsubstanz, Anat. Hefte, 1896, vii, 115.

STOCKARD: I. The Origin of Blood and Vascular Endothelium in Embryos without a Circulation of the Blood and in the Normal Embryo. II. A Study of Wandering Mesenchymal Cells on the Living Yolk-sac and Their Developmental Products: Chromatophoes, Vascular Endothelium and Blood Cells, 1915, Memoirs Wistar Institute of Anatomy and Biology, No. 7.

STRICKLER: Vorlesung über allgemeine und experimentelle Pathologie, 1883, 419.

VALENTIN: Wagners Handwörterbuch der Physiologie, Braunschweig, Vieweg, 1842, i, 670.

VIRCHOW: Ueber die Identität von Knorpel-und Bindegewebskörperchen sowie über Schleimgewebe, Würzberger med. Verhandl., 1852, ii, 150.

WEIDENREICH: Die Leucocyten und verwandte Zellformen, Bergmann, Wiesbaden, 1911.

ZIEGLER: Untersuchungen über pathologische Bindegewebs und Gefässneubildung, Würzburg, Standinger, 1876.

CHAPTER VI

NATURE AND GENESIS OF PERITONEAL ADHESIONS

By adhesions in the sense of this monograph is meant the more or less permanent attachment of two peritoneal surfaces. In general clinical parlance one understands by this, undesirable attachments which follow irritation produced by infection or from some injury. This is an erroneous conception, for adhesions in nearly all instances are like inflammations; salutary processes. Our antipathy should be directed, not against the adhesions, but against those factors which produce them, be these infection or our own misguided efforts.

That adhesions are favorable in their disposition is clear when we recall that the healing of the gut walls in anastomoses is rendered possible because of the readiness with which the peritoneal surfaces about our suture lines unite. Without this facility in healing our operations on the gut tract would be uniformly fatal. These represent permanent adhesions and they follow the general laws of healing by first intention.

Conversely, if adhesions did not promptly attend inflammatory reactions all pathologic perforations would be fatal. It is the facility of peritoneal surfaces to become attached to each other when irritated by reactive processes thus walling in the affected area, that protects the general peritoneal cavity from a diffusion of the infection. The general type of tissue reaction corresponds to what has already been discussed under healing by second intention. Since these adhesions tend to become absorbed after the infection has been conquered they may be called temporary adhesions.

Besides these two general types, permanent and temporary adhesions, it is necessary to note conditions often referred to by clinicians as adhesions which really have nothing to do with the processes above enumerated. These are agglutinations and fetal adhesions. The former represent chemical changes in the peri-

toneal fluids and the latter variations in visceral topography. These conditions may be disposed of first.

I. **Peritoneal Agglutination.**—This term may be applied to the condition of slight attachment noted when some chemical change has occurred in the peritoneal fluid. The difference between the changes and the initial stages of adhesions consists in that in the former the attachment is brought about by coagulation of the fluid normally covering the endothelium unaccompanied by deeper changes. In other words, there is no evidence of irritation manifested by vas-

Fig. 112.—Fine fibrin bridges which appeared after two surfaces were pulled apart after they had become agglutinated.

cular dilatation and exudation, but the process is played in the alteration of the surface secretions normally present. There results fine fibrin bridges between the surfaces of the endothelial cells that look on cross-section like cilia (Fig. 112). With greater irritation the cells may become thickened so that they are cuboid or even columnar. A prolonged exposure to the air may produce these agglutinations. They may be observed in abdominal operations where a dry pack has lain against a gut for a considerable

length of time. The same thing is observed when two serous surfaces lying in contact are exposed to the air. When separating such surfaces with the fingers a sensation is experienced which reminds one of the crackling of an automobile tire while running on a slightly muddy street.

These agglutinations may temporarily hold gut surfaces in light apposition. As soon as the parts are returned to their normal environment the secretion of the serosa increases and the surfaces free themselves. Permanent adhesions do not follow this process because there are none of the active factors which characterize the initial processes of adhesion formation. There is no change in the intercellular substance and no change in the connective tissue of the basement membrane. In order that adhesions shall follow agglutinations some factor must be added which will initiate the fibrin-producing process. A continuation of the same process may do this, as for instance the continued contact of a gauze pack will be followed by the formation of adhesions if the foreign body can be surrounded by peritoneal surfaces.

Perhaps those imperfect attachments of surfaces often seen in grave general infections of the peritoneum may be classed here. One often finds coils of intestines loosely held together by a smeary fluid. In these conditions the surfaces are not held together by any purposive reaction on the part of the tissue but by products normal to the part changed by unfavorable environment or overwhelming intoxication.

The attempt to separate this condition from that of adhesions may seem artificial. Nevertheless, there is a microchemical difference as indicated, and while confusion may be caused by its being associated in some cases with true adhesions, its separation from them is desirable both on scientific and clinical grounds.

The lumping together of agglutinations with adhesions has resulted in attempts to break up these fanciful attachments by the early administration of purgatives and the like. The attempt to force function on an organ the walls of which are paralyzed by the irritation of the operation has resulted in needless discomfort to the patient.

Graser does not distinguish between these simple agglutinations and the early stages of the adhesion processes. He was led to

regard agglutinations as the first change in the formation of adhesions. He made no attempt to distinguish the various factors by tinctorial study nor to follow their ultimate fate.

In order to study the fate of these early changes I sewed windows in the abdominal walls of animals so that the process could be directly observed. By this means it is possible to observe that these agglutinations release themselves within a few hours when left to themselves. An artificial excitation of peristalsis hastens it, as does the excitation of peritoneal exudates.

II. **Prenatal Adhesions.**—In the course of the evolution of the intestines certain attachments of free surfaces take place. Although the attachment follows a general plan, there are variations over rather wide limits. When these exceed the observer's ideas of legitimate anatomic peculiarities they are regarded as abnormal. Since they are obviously congenital and no known cause for their existence can be determined, a clinical term was borrowed and we have the designation "fetal adhesions." In order to complete the analogy they were ascribed to "fetal peritonitis." Flint has discussed them and recognizes them as legitimate deviations from the normal average. The most of these variations have been referred to in the section on anatomy. Clark regards them as potent factors for future trouble. When the common occurrence of unusual folds and attachments is considered and the exceedingly rare occasions when they are associated with pathologic processes, such occurrence may be regarded with propriety as a coincidence. This viewpoint is substantiated by the discussion in the chapter on anatomy.

It is unfortunate that the term "adhesions" should have ever been applied to these conditions, for the term suggests to the surgeon something abnormal and baneful as is exemplified by the recent campaign directed against the variations in the ileocecal region. Except when needed to bolster up a theory they are seldom observed.

Our knowledge of the genesis and clinical consequences of these anatomic peculiarities is at present wholly unsatisfactory. The sole interest in them centers in the determination of the method pursued in the obliteration of serous surfaces in development. When these occur, variations are established in the embryo as in

the fixation of the cecum to the parietal peritoneum; the serosa, as previously noted, has not reached its full development. The endothelium is low and cuboidal, the basement membrane is imperfectly formed, and the membrana propria is a cellular mass of undifferentiated cells. When in this state a fusion is a simple matter and is in nowise comparable to any process observed in the adult. As already noted in the section on anatomy the causes of this fusion are not understood. Whether pressure of contiguous surfaces has anything to do with it is difficult to say. Fusion can be caused by pressure of two apposed peritoneal surfaces between two metal plates, but this does not prove that pressure has anything to do with the normal process which obtains at the time of development. In the study of the early development of the embryo one is astonished, not at the variability, but at the constancy of the final attachment.

When the whole problem is reviewed from the anatomic, as well as the physiologic standpoint, it must be apparent that there is almost no limit to the range of variability of attachment of the various organs, compatible with physiologic function. The most astonishing variations are not infrequently found in bodies which caused no inconvenience to the individual during life. That they may be associated with diseased conditions, as suggested by Clarke, may be readily accepted, but to ascribe to them categorically all sorts of baneful influences manifesting themselves late in life is obviously apt to lead to serious error. There is no question but that their misinterpretation has caused more suffering than their existence.

III. **Permanent Adhesions.**—The processes incident to the formation of permanent adhesions are identical with those of wound healing; therefore, in order that an adhesion shall be permanent there must be sufficient irritation to produce an exudate capable of forming fibrillar fibrin. Such a process may be set up (1) by irritation from chemical substances, the product of disease or artificially introduced; (2) by disease processes which excite a mild form of reaction; (3) by mechanical violence, due to accidental traumatism or by the misdirected manipulations of the surgeon.

1. *Adhesions Due to Chemical Irritation.*—During the time when antiseptics were employed in the peritoneal cavity this variety

was of frequent occurrence. Iodoform was no doubt the arch enemy. Many instances of intestinal obstruction were recorded against it. Recently iodine has been reintroduced in the form of the tincture. A repetition of the records against iodoform is as yet of the future. The whole list of substances introduced from time to time as a means of preventing adhesions might with greater propriety be recorded here.

Cyst contents are rarely the cause of adhesions. Echinococcic and mucus-containing cysts, and those in which hemorrhage has occurred, may sometimes be the cause of limited adhesions.

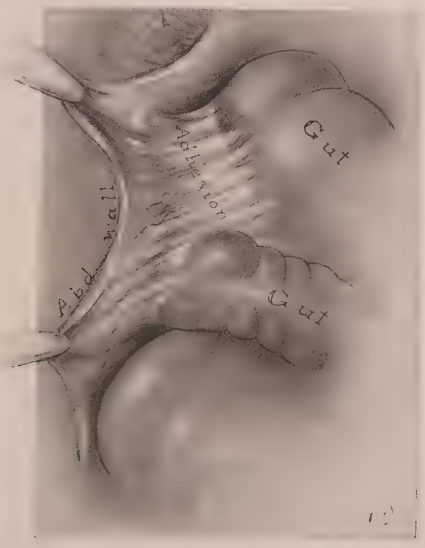

Fig. 113.—Adhesion of the colon to the parietal peritoneum of a rabbit caused by a pledget of gauze placed in the abdominal cavity.

The whole chapter of adhesions due to chemicals exists only because of a too literal acceptance of Hunter's dictum, "Don't think, try." Judging from the history of surgical technic, this is the most pernicious advice ever given to a surgeon.

2. *Adhesions Due to Disease Processes.*—Adhesions due to disease processes may be produced by any condition which excites the formation of a coagulable exudate on the surface of the peritoneum. This implies that the irritation must be of moderate intensity, for most disease processes excite an irritation in excess of this degree

and produce an exudate which forms a granular instead of a fibrillar fibrin, hence only temporary adhesions result.

The genesis of these conditions can be imitated by experiment. If a pledget of gauze or other suitable unabsorbable body is placed in the peritoneal cavity, it at once causes adhesions to form about it (Fig. 113). The irritation produced by an extraperitoneal disease is most nearly imitated by placing a gauze pack against the pari-

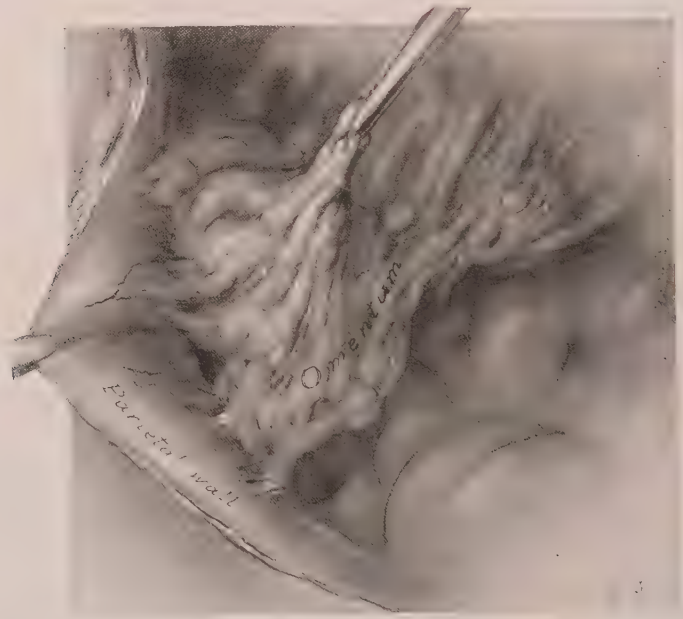

Fig. 114.—Omentum adherent to the abdominal wall, the result of aseptic peritoneal irritation produced by placing a pledget of gauze over the parietal peritoneum and under the abdominal muscles without opening into the abdominal cavity.

etal peritoneum without opening the abdominal cavity. The gauze causes a reactive inflammation in the parietal peritoneum, just as a diseased process would. This reaction is transmitted to such abdominal viscera as may be lying near the point of irritation, and causes their adhesion to it (Fig. 114). This experiment imitates very closely an inflammation of the gall bladder due to the presence of stones. The stone irritates the gall bladder wall setting up a reaction which is, in turn, transmitted to the adjoining peritoneal surfaces, and causes their adhesion to it and to each other.

If the foreign body in the experiment is infected by soaking it in a culture of pyogenic bacteria or in intestinal contents, an ulcerous process is excited. This may be so great as to cause the foreign body to be extruded into the lumen of the gut (Fig. 115). It does not require a heavy tax on the imagination to see the protruding gauze pledget in the figure as an enterolith or a gall stone escaping from the infected cavity in which it lay.

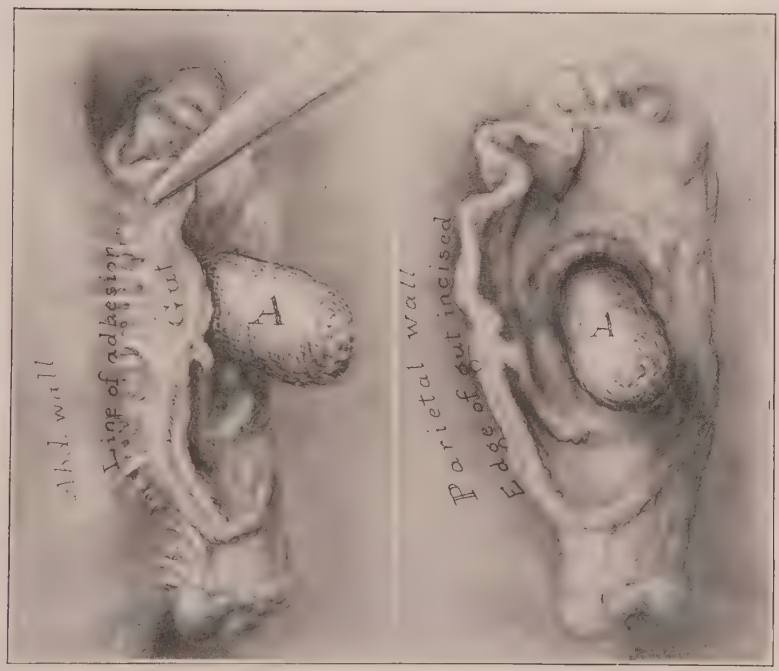

Fig. 115.—A pledget of gauze was placed loose in the abdominal cavity. The gut wall which has encapsulated it has become ulcerated through, and the gauze appears in the lumen of the gut. *1* shows the density of the adhesions of the gut to the parietal wall; *2* shows the gut wall split open to show the protruding gauze.

The chief interest centers in the discovery of those conditions which determine the permanency of the adhesions. In general terms, it may be said that, in order that the adhesion shall be permanent the reaction must not have been sufficiently great to prevent the formation of fibrillar fibrin. This generally means that there is a great irritation, as from the escape of pus-producing organisms, the immediate region of the more intense infection is

Fig. 116.—Residue of adhesions remaining after an attack of appendicitis. The colon is attached to the parietal wall, the omentum is adherent to the colon, liver and anterior abdominal wall.

surrounded by temporary adhesions, while, at some distance from this point where the infection is less intense, permanent adhesions may form. The interpretation of these past processes may cause some confusion. For instance, in an appendix with the chief site of irritation at its base, massive omental adhesions occur at the height of the process, which are temporary in character. At some distance from this, say at the level of the lower pole of the kidney, the irritation may be just sufficient to produce a plastic exudate. At this point, therefore, remote from the chief site of infection, the adhesion is permanent (Fig. 116).

The relation of permanent adhesions to the more intense infections may be studied in the case of the foreign body experiment represented in Fig. 115. At first over the site of the foreign body an exudate of fibrin is thrown out. This being invaded by bacteria can not produce fibrous tissue. The irritation produces more exudate farther away from the maximum irritation which at some point is sufficiently infiltrated with defensive factors to wall in the infection. External to this zone a layer of fibrin is then thrown out, which being protected from the infection by the protecting wall, is able to form fibrous tissue. As a final result the central zone above mentioned, on account of the intensity of the infection, liquefies, destroying the wall of the gut, and a perforation ensues, but a general peritoneal infection is prevented by the more remote zones of adhesion. The outermost of these zones becomes a permanent fibrous tissue union. If a permanent fistula results a fibrous tube covered by peritoneum remains. If the fistula heals a fibrous cord covered only by peritoneum remains as a permanent reminder of a serious condition successfully withstood. These zones may be represented by a schematic drawing (Fig. 117).

The various steps represented in the above experiment can be traced with variations in impending perforations of many disease processes, for instance in enteroliths or gall stones. Instead of the perforation being direct into a hollow viscus, complications, such as the formation of abscesses and secondary complications, due to these abscesses, may ensue. In fact, in most instances, not a fistula but a localized abscess is the immediate result. If the process stops short of a perforation the first step in the process only may be reached, as represented in Fig. 118.

Similar processes can be traced in more complex conditions, such as are observed over chronic ulcers, as of the stomach and duodenum. The various possibilities may be outlined as follows: immediately over the ulcer the reaction, if mild, excites the peritoneum to the formation of permanent adhesions. In this way the lesion is permanently patched, and disaster averted. If the reaction is greater, granular fibrin immediately forms over the ulcerous area (Fig. 117), and it is only some distance from the site

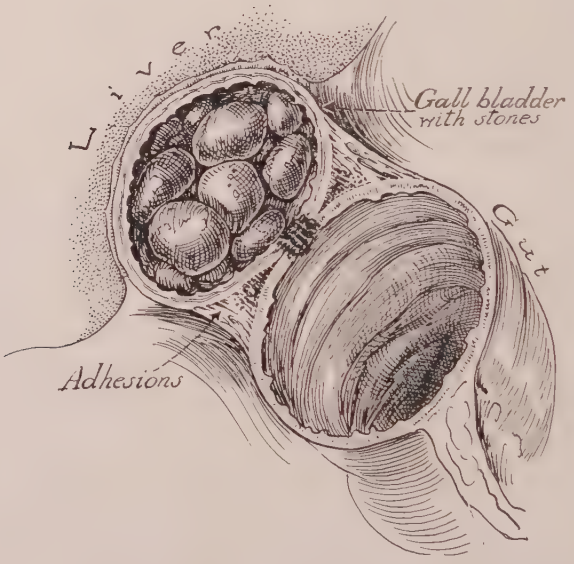

Fig. 117.—Schematic drawing showing the relation of temporary adhesions in gut perforations. The middle dark zone between the hollow organs represents the portion of the wall undergoing necrosis. The granular zones at the side of it represent temporary adhesions walling in the necrotic area, while the outer striated zones represent fibrillar fibrin, which will form the permanent adhesions.

of greatest irritation that permanent adhesions can form. If the progress now stops as an end result we see a permanent adhesion some distance from the scar of the lesion, while over the lesion itself the temporary adhesions have been absorbed and the peritoneal surface is free from adhesions. How this process operates can be seen by observing the end results of many old perforations. For instance an impending duodenal perforation may leave its mark between the liver and the diaphragm, between the

Fig. 118.—Adhesions between the omentum and the parietal wall. There are also extensive adhesions about the gall bladder, liver, and abdominal wall. A pyloric ulcer which had healed was the cause of the infection.

stomach and the colon, between the omentum and the colon or the stomach, etc. It is not unusual to find adhesions some distance

from an old scar in the region of the outlet of the stomach. For instance, adhesions between the gall bladder and the colon and a dimpled scar near the pylorus may be all that remains of a process that once saved the patient from an impending perforation.

To convince oneself of the correctness of this interpretation of the sequential processes one must observe the various stages of the condition in a series of patients. I once operated on a patient who gave a history typical of perforation of a gastric ulcer. After some weeks he recovered, and was fairly free from disturbance until a day before operation when renewed symptoms developed. At operation young adhesions quite similar to those illustrated

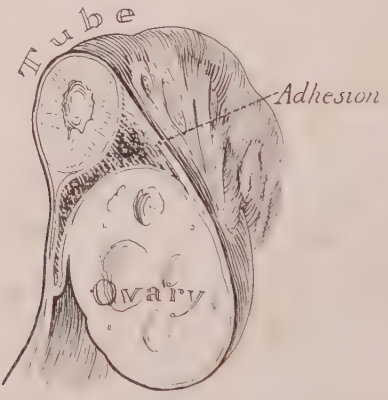

Fig. 119.—Schematic drawing of an infected tube and ovary. The granular area between the tube and the ovary represents the temporary adhesion, while the longitudinal lines above it represent the fibrillar fibrin, which forms the permanent adhesions after the granular material beneath has become aborted, forming a free membranous adhesion. (Compare Fig. 120.)

in Fig. 118 were found. An old duodenal ulcer was adherent to the gall bladder and colon, and was thus partially walled off. A secondary perforation had taken place, giving rise to renewed symptoms. The ulcer was resected with as little disturbance of the adhesions as possible.

More striking still are those instances in which actual escape of gastric contents occurs without preliminary adhesions. The extent to which these adhesions may form is astonishing. In one autopsy which I viewed there were adhesions between liver and abdominal wall; colon and gall bladder; omentum and colon were

attached to the abdominal wall (Fig. 118). A healed ulcer of the pyloric end of the stomach showed a puckered scar of the peritoneum, itself free from adhesions alone remained to reveal the site of a healed ulcer of the pyloric end of the stomach that had caused the remarkable adhesions.

The relation of the healed ulcer to the adhesions depicted in Fig. 118 can hardly be doubted. Material escaping from a perforation in the duodenum tends to gravitate downward over the watershed furnished by the colon and omentum. Fluid is early found laterally to the colon and cecum in such cases. The first patient I

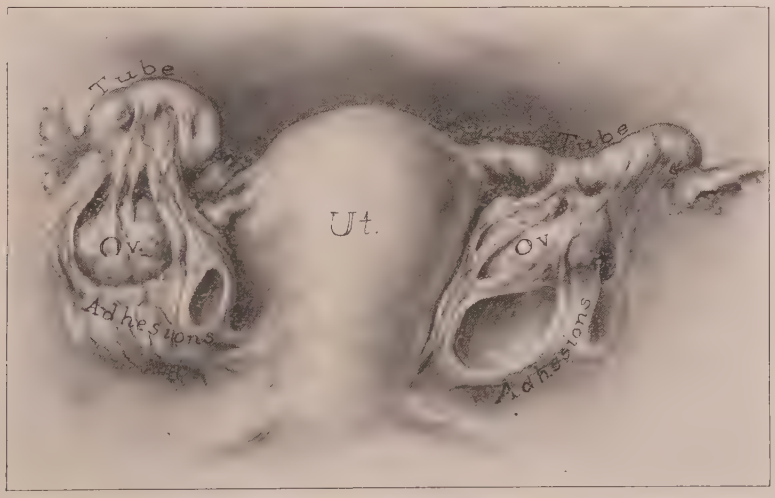

Fig. 120.—Permanent adhesion bands about the tubes and ovaries after a gonorrheal salpingitis. This represents the end stage schematically represented in Fig. 119.

ever operated on for perforation of a gastric ulcer presented strands of sauerkraut distributed from the point of perforation over the colon and laterally to it and over the right half of the great omentum. The inflammation was confined to this region and to the subhepatic region. So universal in fact has been the observation of this tendency of the fluid escaping from perforations that pelvic drainage is regularly instituted. Operations on the second or third day sometimes reveal early adhesions. True, not all surgeons agree with this interpretation. Pilcher quotes a case in which similar adhesions were encountered. He thought the infection

began in the region of the appendix and extended upward. This explanation, I believe, goes counter to the experience of most surgeons. This is certainly true in those cases in which there is a healed perforation in the stomach or duodenum, indicating a former source of infection. Similar cases are recorded in the literature. Riedel records a case in which, following a gastric ulcer, the duodenum, stomach, and great omentum were matted together.

Such adhesions can occur only in cases where the infective material escaping from the stomach is of but slight virulence. This must be admitted because the patient recovers spontaneously—not a very frequent termination at best. In the more virulent cases, rescued only by operation, the infection may be so virulent that no permanent adhesions result except about the regions where the gauze drain is placed, when such is used.

These permanent adhesions are most frequent in the pelvis following gonorrheal infections of the tubes and may most easily be studied there. The gonococcus is a pronounced fibrin-producer, and the permanence of the adhesions is due to its relatively mild pathogenicity. An enormous fibrinous exudate, both beneath the peritoneum and on its surface, is produced. That beneath the peritoneum is granular and later undergoes absorption and the major portion of that exuded upon the surface of the peritoneum is of the same character. On the surface of this granular exudate a layer of fibrillar fibrin tends to form. This is particularly true in the milder infections or in those of long duration.

Here the general plan of pseudoperitoneal formation can be studied best. An exudate about infected tubes and ovaries fills in the chinks between the neighboring intestines. This is granular fibrin and receives on its surface a fibrillar layer (Fig. 119) which bridges the gaps between ovaries and tubes with bundles of fibrin. When this process has finally run its course the granular fibrin filling the chinks is absorbed, while the surface layer becomes organized into fibers. There remains only the new membrane extending across the space from the tube to the ovary (Fig. 120). These adhesions may be very thin, and sometimes are interrupted—a condition designated in clinical parlance as "cobweb" adhesions, with which every surgeon's fingers are familiar; or they may form bands or even form complete membranes totally encapsulating the ovaries.

3. *Adhesions Due to External Violence.*—Adhesions may result from contusion of either the parietal or the visceral peritoneum, or of both. The process may be imitated by exposing the transversalis fascia, pinching it and the peritoneum with forceps. Adhesions will form at this point. Opening the abdomen and crushing any internal organ produces the same result. The necessary factor is that the trauma must be sufficient to produce the exudate, yet not great enough to start processes which will destroy the coagulability of the exudate.

Blows by blunt objects may injure a hollow organ to such a degree that necrosis of the traumatized area occurs. If the process is slow, adhesions may cover it in part. An instance of this may be cited as follows: A man was kicked in the abdomen by a horse and after three weeks a fecal phlegmon formed, which, when drained, left a fistula extending to the gut. A later operation showed a fibrous tube formed by permanent adhesions leading from the gut to the exterior of the body. In this case the center of the lesion was injured to such a degree that permanent adhesions could not form, while adhesions did form at the periphery. This process may be reversed when an infection becomes walled off in the peritoneal cavity. Here the infective mass gradually destroys the wall of an adjoining hollow viscus until perforation is complete and the abscess empties itself into the lumen of the gut. If the process is of low virulence, and encapsulating adhesions become fibrous, a permanent adhesion results, perchance, some distance from the original injury.

Another case may be cited to exemplify the greater degree of injury. A carpenter fell from a scaffolding striking his abdomen on a blunt object. After some hours he returned to work. On the fifth day he suddenly began to suffer from a generalized peritonitis. At operation an opening in the gut the size of a dime was found, due to the necrotic separation of the contused area. Here the tissue was so badly injured that even a temporary adhesion did not form, and when the contused area separated by necrosis, general infection of the peritoneum followed.

4. *Adhesions Due to Injuries During Operations.*—The most fruitful source of permanent adhesions is the manipulations of the surgeon. Any injury to the peritoneum produces an exudate capable

of forming an adhesion. Incision into the peritoneum has been observed in the human subject with scientific exactness. When a patient, the subject of a previous operation, is laparotomized a second time it is very common to find the omentum adherent to the site of the former incision. In the days of the through-and-through suture, which allowed the edges of the peritoneum to retract and thus to become exposed to the abdominal contents, adhesions were almost universal. As our technic improved, the effect of more careful closure of the peritoneum became manifest. It has been found that, if the edges are carefully everted into the wound, allowing only a suture line to present toward the peritoneal cavity, adhesions are prevented with a considerable degree of certainty. These observations are sufficient to demonstrate the importance of the cut edge of the peritoneum in inviting adhesions. Other injuries, such as perforations by the teeth of forceps, or marked blunt trauma inflicted by retraction, act in like manner. Pedicles remaining after the ligation of peritoneal-covered organs, such as the appendix or a portion of the omentum, leave exposed surfaces which are almost certain to result in adhesions. Any foreign body allowed to remain in the peritoneal cavity results in adhesions.

While such obviously gross lesions result in adhesions almost with certainty, much speculation has been indulged in as to the influence of minor factors. The problem of interest here is the determination of the degree of injury incident to operations which will initiate the process of adhesion formation. It may be set down as a fundamental law that the loss of endothelial surface will produce sufficient exudate to secure adhesions. The difficulty lies in determining when this degree of injury has been inflicted. Surgeons' estimates of it have varied greatly.

Among the lesser degrees of injury that may produce adhesions may be mentioned mere puncture by the suture, as in inverting an edge of peritoneum. This likelihood is increased if the suture material has been sterilized by a chemical irritating to the peritoneum, notably, iodine. The constriction of a bit of tissue by a ligature causing necrosis of the part so constricted, produces adhesions. Blood clots beneath the peritoneum act in the same way. Corbett demonstrated this simply by puncturing a mesenteric vein

with a needle, allowing a small amount of blood to escape beneath the peritoneum. The effect of blood in the free peritoneal cavity on the formation of adhesions has been variously estimated. Fluid blood, generally speaking, is absorbed without any irritation. Clotted blood when lying on uninjured peritoneum likewise is absorbed without injury. When lying in the region of injured tissue the presence of clots invites adhesions. The reason for this is obvious. When lying on uninjured peritoneum none of the factors which excite to fibrous-tissue formation enter the field, while when there is injury to the surrounding tissue these processes at once become active.

It is safe to say that any manipulation which is capable of destroying the endothelial layer is capable of forming adhesions. It is difficult to say whether or not, as the result of any manipulation, cells have been completely removed, or if the basement membrane has been injured. Obalinski believed that injury from the surgeon's fingers during manipulations of the gut could cause such a degree of injury. Vogel, on the contrary, found that very vigorous rubbing with the gauze-armed finger failed to cause adhesions.

The amount of violence necessary to produce a degree of abrasion sufficient to cause adhesion has been greatly underestimated. Actual abrasion of endothelium requires considerable violence. To secure isolated endothelial cells for study it requires considerable effort with a dissecting needle to loosen them. The peritoneal surface is physiologically more delicate than it is anatomically. For instance, a surface may be rubbed with considerable violence with a gauze-armed finger without destroying its anatomic integrity; the physiologic equilibrium, however, will be upset. The vessels dilate and the exudate begins to show between the cells, as may be shown by staining with silver nitrate solution. Such reaction is usually not followed by adhesions, however, since the reaction ceases with the removal of the irritant. The omentum is much more sensitive to mechanical insults than other endothelial covered surfaces since its perivascular tissue is more sparse and the capacity to return to normal is much lessened in consequence.

On the other hand, if the entire basement membrane, with its connective tissue, is removed, much of the adhesion-forming power is destroyed. The underlying muscle makes very feeble efforts at

forming adhesions as we well know from experience in making anastomosis of the esophagus and extraperitoneal portion of the large gut. This may be easily demonstrated by dissecting off the parietal peritoneum or by destroying it with a cautery. We make practical use of this quality when we cauterize, with the actual cautery, such surfaces which can not be covered with peritoneum.

Simple pressure without puncture may produce adhesions as can be demonstrated by retaining two peritoneal surfaces in close apposition by means of lead plates. The Murphy button depends on this quality to secure union of the ends of guts. Fecal impaction in rare instances may act in the same way. The pressure need only be strong enough to shut off the circulation. Death of tissue then occurs, and the dying tissue produces the irritation which initiates the fibrin-forming processes.

A great variety of chemicals are capable of injuring the endothelium to a degree sufficient to cause adhesions. All of the antiseptics cause it, iodine being perhaps the most active. Uyeno found that even weak iodine solutions caused adhesions. The diffusion from iodized catgut when used in the peritoneum may excite to extensive adhesion-formation.. Corrosive chemicals are less likely to cause adhesions, because the basement membrane is destroyed. Strong solutions of iodine may act in this way. Such solutions, instead of exciting the peritoneum to fibrin-formation, precipitate the exudate into a granular debris incapable of fibrilization.

Prone as is the peritoneum to form adhesions, it is doubtful if mere exposure to air can excite adhesions. My window experiments made me believe it does not. Walthard, on the other hand, accused the contact of air with the most dire effects. According to him contact with the air during operation produced adhesions unless the surfaces were protected by moist packs. He believed such contact invited infection also. Uhlmann contradicts these statements.

There are individual variations in the capacity to form adhesions. Constitutional diseases, particularly those of the blood, reduce the capacity to adhesion-formation. This, in a measure, parallels the effect on the coagulation time of the blood. This is quite true for the initial processes and less so for the changes which occur in the

later stages of the healing processes. The factors here involved are too intricate to permit of solution in our present state of knowledge of the chemical processes in diseases of the blood.

An excessive individual predisposition to the production of adhesions is sometimes noted. The following condition is sometimes encountered: an individual who has been operated on develops adhesions and is reoperated on and adhesions reform. This sequence lasts as long as the patient does, or the hopeful persistence of the surgeon endures. Often the disposition to unusual production of adhesions is stimulated by the pernicious habit of "breaking up" adhesions already present. Frayed edges are produced in this way, which furnish the best possible stimulus to the further production of adhesions. By the time this process has been repeated a number of times an astonishing number of adhesions may be present. In some instances, exudates are very extensive, and coagulation and organization seems to take place throughout the mass. In this way veritable keloids are produced.

5. *Deleterious Effects of Permanent Adhesions.*—The purpose of permanent adhesions is the repair of solution of continuity. Intestinal repairs and anastomoses are made secure by the union of the peritoneal surfaces. Without this faculty abdominal surgery would be impossible. At the same time it must be recognized that the misdirected activity of the process may cause trouble. However, it is the undeserved reputation of adhesions for mischief, due to the slanderous conversation of surgeons, rather than to the real mischief they do, which has brought them into ill repute. If the surgeon would view them as scars with the same open mind that he views scars on the skin for instance, a better understanding would soon ensue.

The disturbance of function of a hollow viscus or pain is the usual symptoms produced by the unnatural fixation. Each of these is a surgical rarity. In order that an adhesion shall cause pain it must limit the mobility of a movable organ. Pain produced by adventitious bands is unusual unless disturbance of function results. Usually, such conditions exist only in the mind of the surgeon, and in that of the patient due to the tutelage of the surgeon. Severance of such adhesions does not relieve the difficulty. Failure to relieve by operation must be sought in a wrong diagnosis, and

not in the occurrence of postoperative adhesions. This is not true in all cases, to be sure, but if this dictum were followed, the vast proportion of relaparotomies might be avoided.

In order that it shall disturb the function of a hollow viscus the lumen of it must be narrowed to an extent incompatible with the proper performance of its function. Such a state is manifested by hypertrophy proximal to the site of constriction. If there is no such evidence of disturbance of function the diagnosis must be questioned. The mere fact that a woman is constipated and has an adhesion does not associate constipation and adhesion in a causative relation. Perhaps she is suffering from splanchnic inhibition because her husband has died or ought to die. Loose interpretation of the rules of logic has so vitiated the literature that few valuable data can be collated.

Adventitious bands, the relics of an old adhesion, are the most frequent cause of real mischief. By suddenly looping over such a band, an acute obstruction may be produced.

Adhesions of a loop of gut to an inflamed focus may occur at the height of the reactive process. The occlusion may be enhanced by the edema from the inflammation. These may be spontaneously freed when the reaction is complete.

The literature is replete with evidence that complications may follow an adhesion which is either the result of external injury or of operative procedures. A number of instances may be cited. Vogel reports a number of striking instances. Riedel records a case in which, following an injury produced by a blunt object striking the abdomen, the abdominal wall, the gall bladder, and the omentum were matted together.

Lucas-Championniere reports a case in which adhesions leading to the ileum followed peritonitis after an ovariotomy, one following the drainage of a local abscess. Dahm reports two cases from Olshausen's clinic in which ileus from adhesions following the treatment of stumps of tumors with iodoform took place. Riedel reports a case in which the great omentum had become adherent between the liver and the diaphragm so firmly that it could not be separated and he was obliged to ligate and sever the adhesion. He adds the remark that afterpains are always severe after the ligation of the omentum.

6. *Late Changes in Permanent Adhesions.*—An adhesion represents a scar, and as such, is prone to undergo the changes such tissue usually undergoes. In this experimental evidence and clinical experience are in accord. In order that any scar may contract, uninterrupted healing must occur. Usually adhesions are formed in the region of diseased processes, and consequently healing is not ideal and stretching is the rule. Most surgeons have had a chance to observe a skin scar contract while the fascial scar beneath stretched because of some irritation during the course of

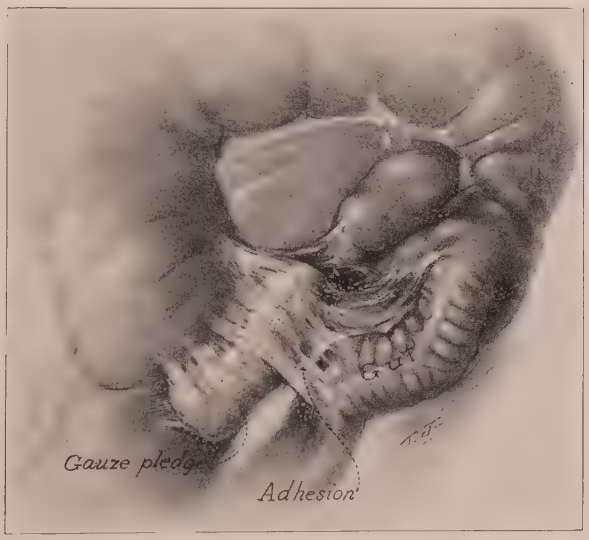

Fig. 121.—Gut wall held close to the foreign body. The foreign body acts as the granular fibrin in the two previous figures. Specimen removed after ten days.

healing. As a rule, during the uninterrupted healing the scar tends to contract, in general, an amount equal to one-third of its length. Traction on a young scar may cause it to elongate. This point can be tested by relaparotomizing animals on which experimental adhesions have formed; and calculations in relaparotomized human subjects will bear this out. When a foreign body placed in the peritoneal cavity first becomes imbedded in adhesions, the organ lying over it is in actual contact (Fig. 121). As the process becomes older the adhesions elongate. As time goes on, these become much attenuated (Fig. 122). After the lapse of

a considerable period (a month or more), the adhesions may be thin and web-like (Fig. 123). Very likely they may separate entirely. This is more certainly true if there is any disturbance in the process of wound healing. This tendency to stretch is most marked in the case of the great omentum. Figs. 124 and 125 show different periods of the same adhesion.

While adhesion formation can be followed accurately and re-

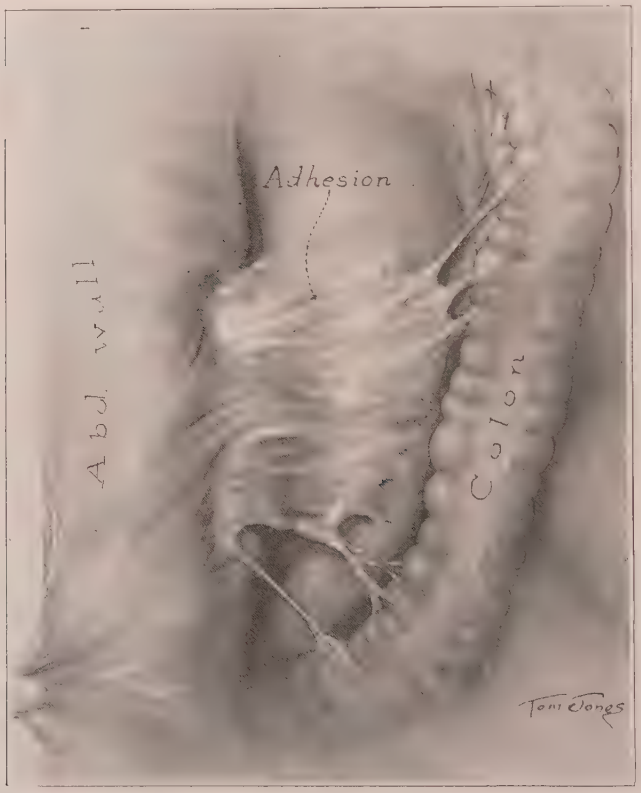

Fig. 122.—Adhesion as in previous figure showing beginning attenuation due to traction.

sults predicted in animal experimentation, speculation must always enter in the human subject. Attending the adhesion formation during diseased processes there is always some disturbance to the ideal condition for fibrous-tissue formation. The reaction caused by the disease influences the fibrin formation. The process is re-

tarded. Often many weeks are required for the process of change from fibrin to fibrous tissue to occur. Usually there is intense cellular infiltration and an extensive serous exudate.

Adhesions formed under such conditions, like any scar formed in an infected environment, are prone to stretch. In any human subject in the drainage of abscesses, intestinal coils may be observed to be held in contact by adhesions. A later operation may show only an attenuated band holding these coils together. I have noted

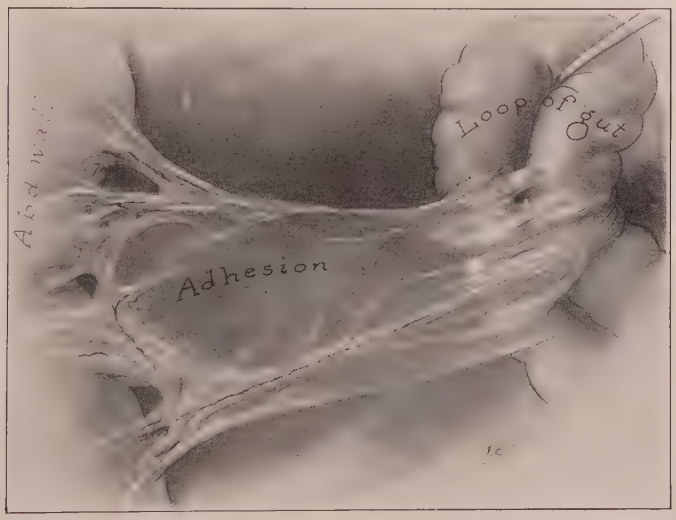

Fig. 123.—A still older adhesion showing more pronounced attenuation.

this most frequently in conditions which resulted in fecal fistulæ requiring a secondary operation to close.

It will be readily recognized that in this respect these adhesions behave quite like scars formed in an infected abdominal incision. Here it is well known, other conditions aside from frank infections may cause a corresponding disposition to stretch. Injury to a parenchymatous organ, including muscle, may produce unfavorable conditions in healing in adjacent fibrous tissue resulting in a disposition of the resulting scar to stretch.

When an infective process is continuous, masses of scar tissue may be produced. This is sometimes seen in diverticulitis, caus-

ing an extensive cicatricial thickening of the mesentery. It is prone to occur also when there is a retroperitoneal infection. I have noted it most frequently in the parietal peritoneum over a kidney when an infected perinephritis has long been drained. Here the scar in the peritoneum over the kidney extending over the duodenum may be represented by a heavy cicatricial mass. The same thing

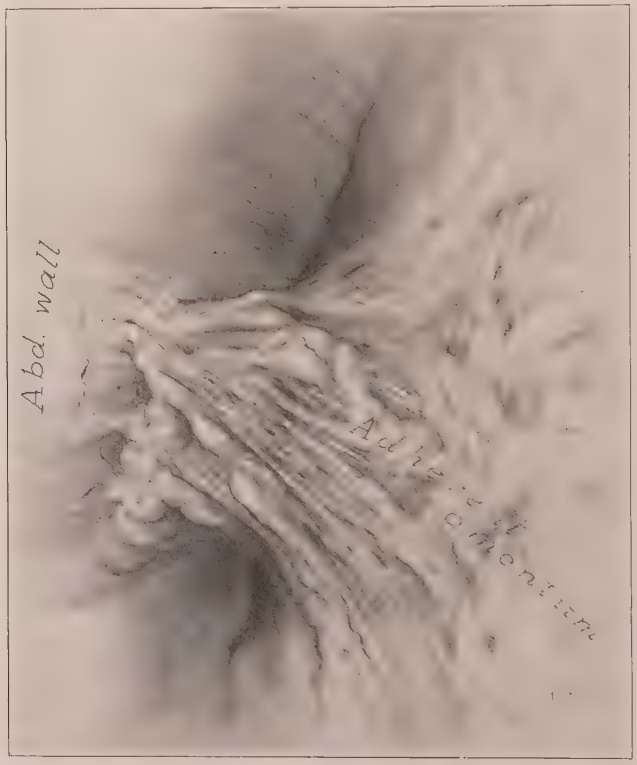

Fig. 124.—Mass adhesion of the omentum to the abdominal incision. Duration, two weeks.

may occur within the broad ligament in parametritis producing retraction on the uterus. The peritoneum may come in such cases to resemble the pleura in suppurative pleurisy.

That an adhesion may become stronger because of continuous traction, as assumed by Lane, is contrary to the known behavior of scars. Since no evidence has been produced to substantiate such

a theory it may be regarded as a peculiar "crystallization of forces" not generally applicable.

7. *Management of Permanent Adhesions.*—Permanent adhesions require attention only when they limit the movement of a mobile organ or contract the lumen of a hollow one.

A number of procedures have been practiced to achieve this end. Massage, particularly of the pelvic organs, once was in vogue but

Fig. 125.—The same adhesion as in Fig. 124 reexamined after four weeks, by a relaparotomy.

the results obtained were negligible. Fibrolysin was employed under the theory that it had the power to dissolve fibrous tissue. There is no evidence that any results were obtained.

When an adhesion exerts a pernicious influence it can be managed only by means of an operation. The offending band must be severed and means instituted to prevent the reformation of the adhesion. A detailed discussion of this subject is given elsewhere.

IV. **Temporary Adhesions.**—In the preceding section permanent adhesions, having for their purpose the repair of solutions of continuity, were discussed. In this section, in contrast with these, temporary adhesions, which have for their purpose the mechanical limitation of infective processes, will receive consideration. This designation is warranted because their duration is synchronous with the duration of the infection. This statement is true in general terms only, for when an infective process is of long duration the induced adhesion may become permanent.

The mechanical limitation of infections is secured by the building of a barrier about the focus of irritation by the union of adjacent organs. This is initiated by the formation of an exudate on the peritoneal surface of the organs which harbor the focus of infection. This exudate, coming in contact with other peritoneal surfaces, excites a similar exudate. These exudates become confluent, and, together with the peritoneal surfaces which produce them, form a barrier to the advancing infection. These barrier walls limit the area of infection that the individual may not be overwhelmed by the absorption of toxic products. The actual inhibition and annihilation of the noxious agents is the task of other agents. It is worthy of note in this place that it is through the barrier walls that these agents reach the site of infection, so that their existence is of more than mechanical importance, though it is this phase only that interests us here.

If these conservative processes are competent, the infected area becomes isolated from the surrounding peritoneal cavity by the barrier wall, and an encapsulated abscess, in clinical parlance, results. If not wholly competent, the infection may spread in one direction while barred in others. On the other hand, infection even when once localized, because of increased tension within the abscess, may break the barrier and spread again. The advancing border may be secondarily limited by new adhesions. This is exemplified in appendiceal abscesses, where a barrier is formed above and medially, but the infection spreads into the pelvis only to be again limited by new adhesions at the advancing border. Because of this tendency to advance, every possible degree of inflammation, from completely localized to the unopposed diffuse, may be encountered.

The pathogenesis of temporary adhesions is altogether analogous

to what has already been discussed in the chapter on the formation of fibrous tissue, as healing by second intention. The exudate, which forms in response to the stimulus exerted by the invading infection undergoes coagulation. This coagulation because of the toxic influence exercised by the infective agents, does not precipitate a fibrillar fibrin, as it does when a permanent adhesion is the result, instead, a trabecular and a granular sort is thrown down. The character of this fibrin is determined by the intensity of the infection, and conversely the firmness of the barrier wall is determined by the character of the fibrin. In intense infections in which the formation of any sort of fibrin is largely interfered with, the adhesions may be absent, or are so feeble that they are powerless to limit the spread of the exudate.

The groundwork of these exudates is a meshwork of fibrin fibers arranged much like the trabeculæ of reticular tissue (Fig. 126). They react specifically with the Mallory and Van Gieson stains but stain imperfectly though perceptibly with Weigert's stain. The less intense the infection, the more complete is this meshwork, and the more typically it stains. These meshes are filled with a granular exudate of varying density. In some instances, however, the meshes are filled with an exudate for the most part amorphous, containing few granules. The granules of this exudate may be regarded as fibrin, since it takes the fibrin stain, though in the more intense infections the histochemical response to specific fibrin stains may be incomplete and imperfect, and in the extremely intense infections may be lacking entirely, and the material present may consist of a debris irresponsive to any stain (Fig. 127). The essential fibrin character of this material must then be hypothecated because of its physical characteristics and because every degree of tinctorial variation is encountered, varying from the typical to the entirely negative. This is true not only in material obtained from successive patients representing variable degrees of virulence of infection, but even in the same patient in different regions and in varying stages of the disease.

Aside from this trabecular and granular material which forms the mechanical factor of the limitation of the infection, there are many cells present, chiefly polynuclear leucocytes, in varying degrees of intensity. In moderate infections they are abundant and their

nuclei and protoplasm are well preserved for the most part. When the infection is more intense the nuclei are paler, the protoplasm is in part destroyed, and, in general the cells are less abundant. With more intense infections the cells may be partly disintegrated, or even wholly so, and the badly staining nuclei lie bare of any protoplasm. In the most intense infections there may be nearly an entire absence of cellular elements, and the exudate is a formless granular mass wholly refusing any stain, so that it is impossible to tell whether the granules represent fibrin or disintegrated cells.

This varying picture may be further complicated by a change in

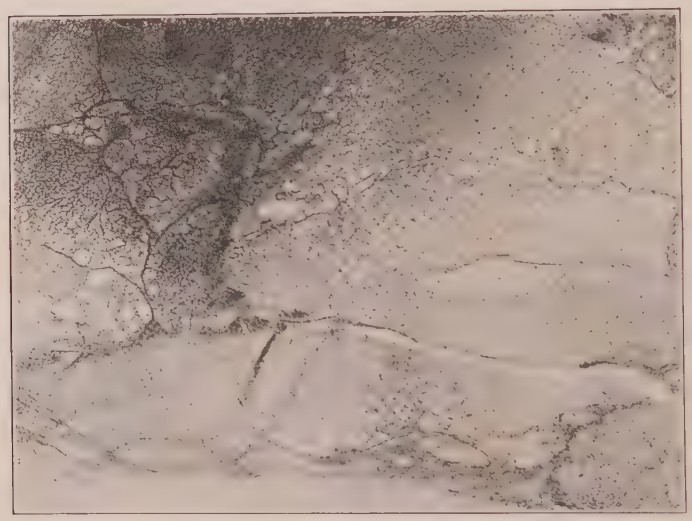

Fig. 126.—A section of the fibrinous exudate in a case of acute appendicitis. The fibrin bundles assume a trabecular arrangement.

virulence during the course of an infection. An infection, moderate in degree at the onset, and stimulating an abundant polynuclear exudate, may cause these cells to degenerate when the infection becomes more intense. In such instances the microscopic field shows a mottling due to the intermingling of the degenerated and the active field.

With the subsidence of the virulence of the infection the contrary changes in cellular contents take place. Among the degenerated cells new deeply staining ones appear. After the infection is definitely regressive, as after the emptying of an abscess, re-

storative processes are particularly well marked. This is marked by the appearance of mononuclear leucocytes. These changes indicate whether the disease is progressing or subsiding. When patches of viable and degenerated leucocytes coexist, an advancing condition may be assumed, while if degenerating leucocytes are intermingled with mononuclear cells, the disease may be assumed to be regressive.

Upon the appearance of the mononuclears, the exudate begins to

Fig. 127.—Granular exudate in which the debris fails to respond to the stains. Many fragments of nuclei are shown.

lessen. The granules are englobed by these large cells and gradually disappear. Whether convoyed out of the region of infection as solid particles or liquefied and absorbed as a fluid substance can not be stated. A search in the neighboring lymph glands for particle-bearing cells has not given trustworthy positive evidence. Some of these cells remain in the infected tissue, become smaller, and their nuclei take the stain more intensely. As this process

continues, the granular debris decreases and the adherent tissues loosen and finally become free.

Within the meshes of the surrounding tissues these large cells may be associated with an increase in the fibrous tissue, and one may follow the typical process of wound healing and fibrous-tissue formation as described by the older pathologists.

When there is great delay in resolution of the infected area, as when an abscess fails to be emptied, either by the energies of the surgeon or by the burrowing of the process, a long-continued absorptive activity of the surrounding tissues is developed. At some distance from the primary site of infection the secondary formation of fibrillar fibrin and the subsequent development of fibrous tissue occurs and permanent union between the adjacent organs takes place. Familiarity with these cellular changes will enable the surgeon to determine whether a given adhesion will loosen of its own accord after the period of its usefulness shall have passed, or whether a permanent union shall be formed.

The foregoing presents in a brief way the cellular factors involved in the formation of temporary adhesions without entering into details about which there has been so much controversy. The cellular phase is more fully discussed in the section on wound healing, and in that on inflammation.

At this time we are concerned only with those inhibiting factors which have to do with the limitations of acute processes. These factors can be understood best by following the stages in a specific region. The principle in all is the same. The appendix may be selected because of the frequency of the opportunity for observing conditions in this region.

When the appendix becomes inflamed a varying degree of subserous exudate forms. The greater the exudate the more likely is the infected area to become walled off. Simultaneously with this exudate in the wall of the appendix, the mesoappendix becomes infiltrated, increasing its thickness sometimes many fold. Not infrequently one-third of the circumference of the appendix becomes enveloped within the swollen meson (Fig. 128, No. 1).

The irritation becomes transmitted from the meson to neighboring structures. The head of the cecum and terminal ileum become thickened, thus aiding in the creation of a barrier. The walls of

other gut loops, which chance to be in this neighborhood, suffer augmentation, and add their bulk toward the creation of a barrier. The posterior parietal peritoneum becomes infiltrated, and adapts itself to the inflamed organ. This is sometimes so marked that a considerable portion of the circumference of the inflamed organ is surrounded.

Aside from the increase in size of the surrounding organs, the most potent factor in the creation of a barrier is the increasing resistance to pressure that these infiltrated walls present. The fingers of the surgeon perceive the extent and character of the wall by the density of the tissue. The appendix may feel like a rigid cord and the gut wall becomes hard and leathery.

The chief organ engaged in the walling-off process is the omentum. It appears at the scene of trouble with a much thickened border. It attaches itself to the inflamed organ and to the surrounding organs which have responded to the inflammation (Fig. 128, No. 2). In this way not infrequently the infected area is covered as with a lid by the thickened omentum (Fig. 128, No. 3). Its heavy bulk adds very materially to the blocking-off process. Like other organs it becomes very dense to the touch, thus aiding materially in securing an effective dam against the spread of the infection.

To these mechanical factors due to the increase in bulk of the surrounding structures, is added the exudate which pours between these organs (Fig. 129). This exudate is precipitated within the chinks and crevices and completes the wall, much as cement acts when cast between crushed rock in concrete structure. Because of the thickening of the walls and mesenteries a very small amount of exudate is sufficient to fill the interstices and to produce their cohesion.

It is striking to note that the exudate within the walls of an organ undergoes the same changes as take place in the exudate on the surface of the organ (Fig. 130). The granular or mesh-like arrangement of the fibrin and the character of the cells, are the same. The only difference is one of accident of topography. This is particularly true of the great omentum, but strikingly pronounced in any subserous tissue.

It should be clearly understood that these processes are conservative and, generally speaking, are evidence that the protective fac-

tors are in the ascendency. The subsequent course depends on the virulence of the infection and other complex biochemical conditions. If the infection is mild, the entire process may regress, leaving no trace of previous disease, or an abscess forms which may subsequently be absorbed or break into the gut or be drained ex-

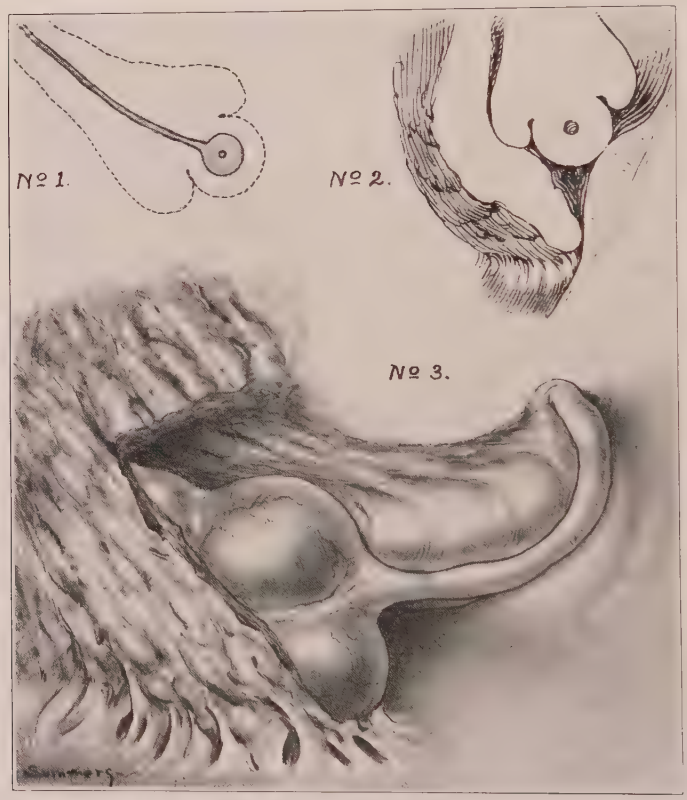

Fig. 128.—*1*. Cross section of the appendix and meson showing the edema of the meson tends to surround the inflamed organ; *2* shows the effect of the edema of the parietal peritoneum and the walling-in by the omentum; *3* represents the same point showing a portion of the omentum cut away to show the thickened tissues beneath.

ternally by the surgeon. In either case the irritation subsides, and the absorption of the exudate begins. The gut walls and the omentum reduce in volume, their density becomes less, and they are once more supple. This process of absorption takes place simultaneously within the interstices and on the surface of the peri-

toneum. The organs which were united become loosened and the adhesion disappears.

1. *Causes of the Migration of the Omentum.*—So marked is the tendency of the omentum to appear at the site of infection that observers have been enthused into the use of poetic expression thereon. Morison referred to it as the "policeman of the abdomen." This,

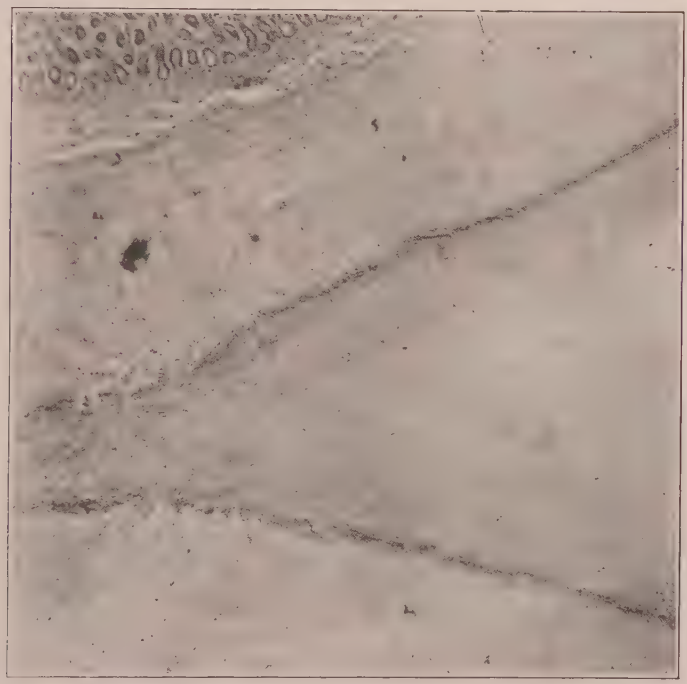

Fig. 129.—The angles between the walls of two apposing guts is shown filled with granular fibrin which holds them in a temporary union.

like most poetic flights, may bear truth without, however, carrying an explanation.

The answer to the question why the omentum seeks the site of infection is not easy. But little has been written upon this point worthy of record. Adami complains of the paucity of the literature. Since that date a number of papers have appeared which present some speculations on this subject. Fisher believes that peristalsis accounts for the movements of the omentum. When inflammation hampers the movements of the intestine the more actively moving

peristalsis in the noninflamed areas moves the omentum toward the affected part. Dickinson ascribes its movement to the pumping action of respiration and to peristalsis. Adami observed that in those bedfast, long the victims of disease, the omenta are often dislodged to the region of the colon or above. Gravity and peristalsis may play a role here, but the conditions are wholly different from those which obtain when there is a movement toward a focus of reaction.

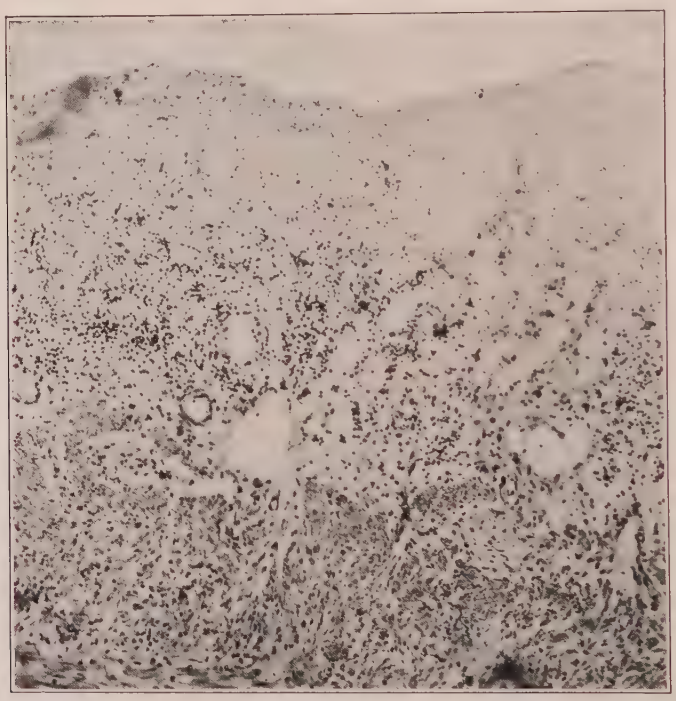

Fig. 130.—Subperitoneal exudate in acute peritonitis. The muscle wall is seen below, the exudate above.

My own studies, while suggestive, are inconclusive. The best that can be done is to trace a similarity of its conduct to that of the leucocytes.

Two methods may be followed in studying this problem. The one consists in the study of the omentum itself under conditions simulating those in disease by directly observing its movements through a window placed in the abdominal wall of an animal; the

other is the study of an artificial omentum under the conditions and environment of the natural one.

Following the first plan, if now a foreign body, as a pledget of sterile gauze, be placed within the abdominal cavity within the range of vision through the window, but away from contact with the omentum itself, the behavior of the latter can be followed. It is noted that an exudate composed of fibrin-forming elements, and leucocytes, form about the foreign body. Quickly following this, an exudate occurs in the meshes of the omentum and on its surface. This accomplished the omentum proceeds toward the foreign body, and the exudate on its surface becomes fused with that about the foreign body. The explanation of these phenomena may be tentatively offered as follows: the fibrinous exudate, and the contained cells, form about the foreign body as a direct result of the irritation. The peritoneum in the neighborhood becomes more or less hyperemic. The vessels of the omentum share this general reaction and respond by a pronounced serous and cellular exudate. The conglomeration of leucocytes is held together by the delicate meshwork of the omentum, and responding to the influence that draws any leucocyte to an area of infection, proceeds to it and incidentally carries the omentum more toward the point of irritation; and since the inflamed omentum is made up largely of leucocytes, it seems reasonable to suppose that the tissue of the organ follows along as an incidental factor. When there is no leucocytic infiltration of cells into the omentum, as in the extremely virulent infections, there is no migration.

Through the window one may study the influence of position, intestinal distention, and peristalsis. The normal omentum is not influenced by position in the presence of normal intraabdominal tension. In the tympanitic abdomen the omentum follows the line of least resistance, but there is no evidence that this influence is a factor capable of propelling it toward the site of disease. By the time the organ becomes sufficiently infiltrated that its weight might be a factor, it has usually become attached to the site of irritation. It remains for some one to suggest that the hyperemia attending the reactive processes increases the weight of the organ, and adds to the disposition to fall toward the affected area. The same may be said of increased peristalsis. The omentum does not

move until infiltrated, and when once infiltrated peristalsis can not move it. It is then so rigid that the intestines glide beneath it. On the other hand, if the intestines become adherent, peristalsis ceases, and they become distended, in greater or less degree.

The rate of migration of the omentum may be studied by means of the x-ray. Small shot may be fastened to the omentum, and a source of irritation set up within the abdomen by placing in a gauze pledget. By continued observations with the x-ray the gradual change in position of the shot may be followed. This is a complicated experiment and is subject to many variations, but, so far as conclusions are warranted from a mean of many experiments, the migration of the omentum may be said to be complete in from four to eight hours with the degree of irritation produced by a gauze pledget.

The other method of study consists in the use of sheets of corn pith placed free in the peritoneal cavity at some distance from a pledget of gauze or in a wound of the peritoneum. The surrounding tissue becomes infiltrated as before. An exudate is also formed in the region of the corn pith. It becomes infiltrated with peritoneal exudate, including an abundance of cells. This mass, consisting of the foreign body and the contained cells, then gradually moves toward the gauze pledget or the wound in the peritoneum. If the gauze pledget of the peritoneal wound causes too little secretion or the corn pith is too far away the migration may not take place. If the corn pith itself excites too great an irritation, it may become attached and therefore be unable to move or it may become imprisoned in a fold of peritoneum. Therefore in performing these experiments many negative results must be expected. A wholesale experiment may be performed by placing many corn discs in the various regions of the peritoneal cavity of a single animal. The incision in the peritoneum is allowed to remain unsutured, closing simply the fascial and skin layers. This forms an active field for the attraction of foreign bodies. When the abdomen is opened some days later, an astonishing number of these discs will be found adherent to the wound in the peritoneum at the site of incision.

Why this migration of omentum and a cell-laden foreign body takes place is as great a mystery as the migration of leucocytes. It is likely a physicochemical problem, of which we are only be-

ginning to gain a faint glimmering. That there is a striking similarity in the response of the omentum to irritation with that manifested by leucocytes, is apparent. If one knows how the leucocytes will respond to a given irritation the conduct of the omentum may be confidently predicted. If we are to speak of the "intelligence" of the omentum we may safely add that an individual's omentum is as smart as his leucocytes. The best answer to those who would give way to outbursts of poetic enthusiasm when contemplating the functions of the omentum, is given by Welch in speaking of the omentum in the following words: "We see here, as elsewhere, that Nature is neither kind nor cruel, but simply obedient to law, and therefore consistent."

2. *Influence of Species of Bacteria on the Character of Adhesions.*—Conditions of the environment and the general state of health of the patient exert a powerful part. Individuals long sick with a disease not involving the peritoneum show a lessened resistance to the spread of any peritoneal infection. This is particularly true in those diseases characterized by a hypoleucocytosis. As familiar examples the perforations in typhoid fever and perforations of the gall bladder in patients who have long been jaundiced, may be mentioned. In each of these the general bodily state is so reduced by disease that the normal leucocytic response to irritation does not follow.

The fact that the infections of the abdomen are seldom the product of a single variety of organism makes the specific characterization uncertain. The gonococcus possesses a great capacity for the excitation of temporary adhesions. The pneumococcus also possesses this characteristic in a marked degree. The staphylococcus in pure culture is prone to produce an entensive degree of infiltration and adhesion formation. The colon bacillus, with its capacity to prevent fibrin formation and to dissolve fibrin when once formed, permits of a less secure barrier formation than the less virulent staphylococcus. The still more virulent streptococcus, with its repellent action toward fibrinous exudate and leucocytic attraction prevents any effort at barrier formation. Various bacteria acting in conjunction or in sequence, may form a bewildering kaleidoscopic picture that fascinates and baffles the mind of the observer. In the pelvis, for instance, the gonococcal perisalpingitis institutes a line

of reaction which may be markedly modified by the appearance of colon bacilli. We then observe the conflict of two varieties of bacteria and the resistant force of the pelvis, each clamoring for the center of the stage.

Not alone do the several varieties of bacteria with their individual characteristics enter to make the process confusing, the ferments of the gut tube add their part. As already noted, perforations which permit the escape of ferments from the gut may dominate the entire picture by preventing the development of the specific characteristics of the bacteria.

The intricacies of intraperitoneal infections and the defensive forces serial and cellular, present a problem upon which there has been but little talent applied. Abandoned by the pathologists and the bacteriologists, and unheeded by the surgeon, this is the barrier across the path of abdominal surgery. There is now a pleasing activity among the anatomists at the present time in the study of tissue reactions and cellular characteristics, and we hope for a securer basis when the study is again taken up.

3. *Deleterious Effects of Temporary Adhesions.*—Not infrequently when adhesions form about infective foci some portion of the gut tract is so unfortunately placed that the lumen is closed. The occlusion is often aggravated by a thickening of the wall of the gut. This state is most often encountered about the gall bladder and appendix. When definite occlusion is produced a temporary fistula must be established proximal to the occlusion.

4. *Late Changes in Temporary Adhesions.*—It may be repeated here that the purpose of temporary adhesions is to prevent the spread of infective processes, and having performed this function they loosen. Simple as the problem is in the abstract, each concrete case requires careful consideration. A few general rules admit of definite statement. When an adhesion has become vascularized and is supple it is apt to be permanent; nevertheless, if there is much traction on an adhesion, the interference with its circulation may gradually lead to its release. If, on the other hand, it is dense in consistency, uniform in color, and brittle in its attachment, it will loosen after the call for its service is ended. If the character of the disease process is kept in mind the ultimate fate of a given adhesion may confidently be predicted. As a general rule it is

safe to say that what is best for the patient is what will likely happen if the adhesion is allowed to remain unmolested.

5. *Management of Temporary Adhesions.*—Temporary adhesions are purposive in character and are brought about in the interest of the patient. Before interfering with them the surgeon should know definitely what he desires to accomplish. Generally speaking, only so much of the adhesion should be broken up as will permit of the accomplishment of the specific purpose for which he is operating. For instance, in acute appendicitis the more direct the surgeon can proceed to the offending organ, and the less he disturbs the protecting adhesions, the less he menaces the patient. If the surgeon attends to the removal of the offending organ, natural processes will loosen the adhesion with much less risk and with greater delicacy than can the surgeon. Cullen has advised the excision of the acute adhesion when possible. In omental adhesions to the appendix, for example, he advises the section of the omentum beyond the site of the infection and the removal of the remaining portion with the appendix. In subacute inflammations this may be safe, but it involves a more extensive operation and deprives the patient of that which once saved him from a general infection. This procedure can be practiced safely only by past-masters in the art of surgery.

Unnecessary interference with adhesions may permit the spread to previously unaffected regions; or, if the infection is mild, a loosened adhesion may become attached to a previously unaffected region and there form a permanent one.

To these possible disasters may be added the inadvertent injury of surrounding structure. These accidents, it is true, haunt the path chiefly of the beginner, but experienced operators sometimes are chagrined at the sudden appearance of mucous membrane.

As a warning to the beginner, it may be pointed out that, in addition to the above-mentioned dangers, it is difficult to determine the limits of adhesions in the acute stage. Normal folds and plicæ, when enlarged by edema, may simulate formidable adhesions, challenging the interference of the surgeon. Yet these, if untouched, retrogress as the infection subsides and the normal is restored, while unwarranted meddling may jeopardize the nutrition of an important organ.

Bibliography

ADAMI: The Great Omentum, Phila. Med. Jour., 1898, i, 373.

CLARK: Surgical Consideration of Congenital and Developmental Defects Leading to Obstinate Constipation, Jour. Am. Med. Assn., 1910, lv, 449.

CORBETT: Peritoneal Adhesions, Surg., Gynec. and Obst., 1917, xxv, 166.

CULLEN: The Surgical Methods of Dealing with Pelvic Infections, Surg., Gynec. and Obst., 1917, xxv, 134.

DAHM: Laparotomie bei Ileus, Halle, a. S. Plötz., 1883.

DICKINSON: The Omentum and Its Functions, Ann. Surg., 1906, xliv, 652.

FISHER: Some Functions of the Omentum, Brit. Med. Jour., 1906, i, 235.

FLINT: Embryonic Bands and Membranes about the Cecum, Bull. Johns Hopkins Hosp., 1912, xxiii, 302.

GRASER: Die erste Verklebung der serösen Haute, Arch. f. klin. Chir., 1895, l, 887.

HERTZLER: Peritoneal Adhesions; Their Cause and Prevention, Tr. West. Surg. Assn., 1904, xiv, 76.

MORISON: Remarks on Some Functions of the Omentum, Brit. Med. Jour., 1906, i, 76.

OBALINSKI: Ueber den Bauchschnitt bei innerem Darmverschluss, Arch. f. klin. Chir., 1889, xxxviii, 249.

PILCHER: A Further Contribution to the study of Pericolic Membranous Films and Bands, Ann. Surg., 1914, lix, 1.

RIEDEL: Ueber Peritonitis chronica non tuberculosa und ihre Folgen: Verengerung des Darmes und Dislocation der rechten Niere, Arch. f. klin. Chir., 1898, lvii, 645.

Ueber adhäsiventzündungen in der Bauchhöhle, Arch. f. klin. Chir., 1894, xlvii, 153.

Die Entfernung von Narbensträngen und Verwachsungen enstanden durch entzündliche Processe in der Gallenblase und in den inneren weiblichen Genitalien, Cor.-Bl. d. allg. ärztl. Ver. v. Thüringen, 1890, xix, 425; 1891, xx, 11.

UHLMANN: Ueber das Auftreten peritonealer Adhäsionen nach Laparotomien, mit besonderer Berücksichtgung des Verhaltnisses zwischen trockener und feuchter Asepsis, Arch. f. Gynaek., 1897, liv, 384.

UYENO: Ueber das Schicksal der peritonealen Adhäsionen und ihre Beeinflussung durch mechanische Massnahmen im Tierexperiment, Beitr. z. klin. Chir., 1909, lxv, 277.

VOGEL: Klinische und Experimentelle Beiträge zur Frage der peritonealen Adhäsionen nach Laparotomien, Deutsch. Ztschr. f. Chir., 1902, lxiii, 296.

WALTHARD: Zur Actiologie peritonealer Adhäsionen nach Laparotomien und deren Verhütung, Cor.-Bl. f. schweiz. Aerzte, 1893, xxiii, 513.

WELCH: Adapation in Pathological Processes, Tr. Cong. Am. Phys. and Surg., 1897, iv, 284.

CHAPTER VII

THE PREVENTION OF ADHESIONS

Adhesions of peritoneal surfaces, as has been noted, may occur to the advantage or the detriment of the patient. The salutary phases have already been considered, and we have here to consider adhesions in the undesirable complications they engender following operation or disease. The prevention of adhesions has given rise to a little study and much speculation most of which has wholly disregarded the fundamental problems of adhesion formation.

In general, there are two classes of adhesions which challenge the skill of the surgeon. The one type forms following a mechanical insult inflicted during the course of an operation or by external violence from a fall or blow. These may be called primary or traumatic. The other type is due to some disease process and represents the end result of some inflammatory condition and may therefore be called the reactive. The relation of disease to complaint is variable. The adhesions may constitute the sole complaint, or the original disease may remain, and the adhesions resulting from it may add a new element. For instance, a cholecystitis may be associated with adhesions. Here there exists the original disease plus the complicating disease. If the gall bladder is drained, a cure may not be effected, and though the original disease may have been relieved, postoperative adhesions may be added to complicate the picture. A careful reading of the literature dealing with adhesions will convince one that the distinction between complications and original disease has been wholly inadequate for purposes of clinical distinction.

Primary Adhesions.—By primary adhesions we may understand those which form after operations in which no adhesions exist at the time of operation. Strictly speaking, there is no such thing as a primary adhesion, but the term suggests to the surgeon a nearness that ought to be salutary if taken to heart.

Adhesions following operations take place at the site of the

incision through the abdominal wall or at the point where the disease exists, as for instance, about the cervical stump in hysterectomy. In the former an adequate closure of the wound insures against adhesion. (See Section on Technic.)

On the other hand, when a temporary denudation is required in order to reach a diseased area beneath the peritoneum, the problem is more complicated. The covering serosa should be preserved, if possible, to cover the area when the removal of the diseased area is completed. This may be typified in transperitoneal operations, as in the removal of the kidney. Here only an incision is required, which is capable of complete closure. Where the removal of the disease requires the removal of a portion of peritoneal surface, sufficient peritoneum must be spared to cover the denuded area. The principle is exactly that followed in amputating an extremity where the first step of the operation consists in making a flap suitable for the covering of the stump of muscle and bone remaining after the operation. This procedure is typified in cholecystectomies and hysterectomies. It is rarely the case that a sufficient peritoneum can not be saved to cover denuded areas in a primary operation, if the surgeon properly plans his operation.

Secondary Adhesions.—By secondary adhesions we may understand those which form during or after a disease has run its course. Usually, as already set forth, adhesions developing in the course of a disease are absorbed as the need for their existence disappears.

Many perfectly normal variations have been regarded as adhesions, the result of some disease. Only a first hand knowledge of anatomic variations can save the surgeon from this error. Examples of the mischief wrought by misinterpretations of normal structures are too recent and too painful for enumeration.

The existence even of an obvious adventitious band is no evidence that it is prejudicial to the welfare of the patient and therefore requires the services of the surgeon. That extensive adhesions may exist without detriment is often seen when operating for other diseases. In general, it is necessary that an adhesion limit the movement of an organ normally movable or seriously constrict the lumen of a hollow organ before its removal is demanded. Where many adhesions exist it is often advantageous to remove only the portion responsible for some disability, leaving the innoxious por-

tion undisturbed. For instance, adhesions may exist about the cecum as the result of a former abscess. The patient may have been wholly free from disturbance after the healing of the abscess until taken suddenly with acute obstruction. At operation a loop of gut may be found constricted by a small band. The severance of this band relieves the condition without the disturbance of other more extensive adhesions. In such instances an attempt to restore original anatomic conditions often leaves denuded surfaces, which will produce greater adhesions than those released. A good rule in such cases is not to disturb what can not be improved.

When Peritoneal Grafts Are Available.—The conditions the surgeon must face at operation are often very complicated. Organs, or parts of organs may be attached to each other, to the abdominal wall, or to the omentum or mesentery, either directly or indirectly, by more or less attenuated adventitious bands.

Unless all denuded surfaces can be covered by peritoneum, reformation of adhesions is liable to result. Unless such a covering can be provided, the surgeon must be sure that the adhesion is distinctly vicious before he severs it.

This is not the place to enter into extensive details of technic, but general plans may be enumerated. Adventitious bands can always be covered by flaps of peritoneum. In that group, in which there is no peritoneum available, a covering must be secured by transplanting a flap from the neighboring peritoneum or from the great omentum. Finally there is a small group in which no covering can be secured, either because there is no tissue available or because the area is inaccessible.

When No Graft Is Available.—It is this class of cases that requires the most careful consideration here. Much experimentation has been done and more written. The logic has been erroneous, and the results pernicious. A small part of the literature is here reviewed with the hope that it may stand out as a blood-written skull and cross-bones for future theorists.

When areas are attached by a very broad base, and no tissue is available for transplant or the parts are inaccessible, the surgeon is obliged to risk the danger of leaving a raw surface within the abdomen, in order to escape a threatening condition. Usually this is the case when adhesions are in inaccessible places. The com-

mon site is in the bottom of the pelvis. When such conditions exist, the surgeon may minimize the danger of re-adhesions by keeping the recently attached surfaces apart. By lifting the uterus or tubes, or both, out of the pelvis re-adhesions may be prevented. The use of rubber protective has long been resorted to by surgeons, and Coffey redirects attention to this means.

Another method which may be useful in rare instances is found in the cautery. Kelterborn developed this method by experimentation on animals, and Webster recommended its use in the human subject. Dembowski held that the cautery invites adhesions. So it may, if only superficial cauterization is done. In order to prevent adhesions, the cautery must destroy the whole basement membrane of the peritoneum.

Another method for prevention of re-formation of adhesions between individual loops of gut is resection of the affected portion. The most frequent condition demanding this procedure is found in the agglutinated convolutions about an old intestinal fistula or about an old wound long drained. It is usually possible in these conditions to resect the entire part of the gut which has been denuded in the separation of the adhesions.

If the surgeon is a man of facile faith he may employ some of the postural measures presented, such, for instance, as the method Tinker recommended, namely, keeping the bowels away from raw surfaces by gravity. Unfortunately, surfaces are usually too closely related to permit their relative positions to be altered by gravity. Any measure to be effective must be employed very early. Clark's method meets this requirement. He uses large hot-water enemata immediately after operation. This method might aid in securing a higher attachment of the sigmoid on the uterus in case a denuded surface were left there, but how it can prevent adhesions is difficult to understand.

A large number of foreign bodies have been introduced into the peritoneal cavity. Time has refuted their claim to usefulness and it is only necessary to record them as matters of historic interest.

Klatz used one to two ounces of castor oil after operation. Byford and Paton recommended the use of physostigmin for the purpose of constantly changing the surfaces. Vogel had some good results and v. Hippel is enthusiastic about its use. Busch and Bibergeil

failed to see results in experimental work. Uyeno likewise saw no results. It may be noted that denuded areas will not glide over each other except by direct mechanical force. Attachment takes place before the nurse can fill the hypodermic for the first dose.

Drugs believed to act specifically on the adhesions have been brought forward. Thiosinamin was tried with negative results by Offergeld in 55 cases. Michael reported good results in 2 cases. Sidorenko denies that the drug exerts any influence on cicatricial tissue. I have tried this drug on skin scars without results. Marvel used adrenalin solution placed directly in the peritoneal cavity. This drug has at least the theoretic advantage of lessening the fibrinous exudate from the peritoneal surface.

Every conceivable foreign body has been employed in the peritoneal cavity, despite the fact that foreign bodies are the most certain means of provoking adhesions.

Of all the foreign bodies used, salt solution is the least objectionable, and the possibility of results the most plausible. Müller and Malcolm recommended this agent. Bainbridge seeks to accomplish the same result by distending the abdomen with oxygen gas before closing the incision. Cleveland inflates the gut with oxygen. Gellhorn advises making the lower part of the trunk hyperemic.

Most pernicious is the use of solid substances. It is not known to whom the blame for starting this procedure should be given. Blake quotes Lathrop as having seen Martin of Berlin rub oil on the denuded surface. Blake found that olive oil did not kill cats, so he employed it in human subjects, not always with good results, it is pleasing to record. Wilkie used it to prevent adhesions and to promote drainage. As a means of prevention of adhesions the supposition is that it forms a uniform coat over the denuded area, which it most certainly does not do. The oil when introduced into the peritoneal cavity soon forms yellowish white caseous-like masses which collect in the folds of the peritoneum. These masses neither prevent adhesions nor invite peritonitis. In my own studies with olive oil I found it always irritated and neither invited nor prevented adhesions. The mass formed from the oil has been referred to as soap though there seems to be no information as to its nature. It irritates the peritoneum and causes a cellular exudate and some

granular fibrin. Schepelmann illustrates some of the effects on the tissues. Furthermore, rabbits in which as little as 2 c.c. of oil was used promptly died. The cause of death is likely a fat embolism of the lungs. At least fat droplets are found in the lungs and the animals die in collapse with dyspnea and cyanosis. The amount likely to be fatal in human beings is not known. Rübsamen saw a fatal collapse after the use of 170 gm. of camphor oil. Vogel found gum arabic useful. Busch and Bibergeil tried lanolin, paraffin, olive oil, and agar, and found that all irritated the peritoneum. Cubbins and Abt found that adhesions occurred when vaseline, paraffin, and olive oil were placed in the peritoneal cavity of dogs in absence of other sources of irritation. A number of authors had good results after olive oil but no one is recommending it now.

Animal membranes have been employed. Cargile (personal communication) seems to have started the practice by suggesting the use of the peritoneum of the ox. Baum used a mat made of catgut and calf's peritoneum preserved in ether and alcohol. Morris recommends the use of shark's peritoneum for the purpose. Craig and Ellis tested out the use of these membranes in a scientific manner and found they encouraged adhesions. Stavely tried out these substances with failure. Lauenstein used silk protective in one case, of course with unfavorable results. One can but join Richardson in the expression of astonishment that no more harm was done by the employment of these substances.

Powdered substances also have been used. It was once the general practice to dust all "raw" surfaces with powdered iodoform. The bulk of the literature on intestinal obstruction following adhesions was published during this period. A close study of this literature leads to the conclusion that it was not the obstruction but the relaparotomies that killed the patients. The distention and vomiting were but the efforts of the peritoneum to negate the insult. Left to itself this would have been accomplished. It is curious that only human material was used for these experiments.

The rule is universal that what the peritoneum can not absorb it walls off or dies in the attempt. Any foreign body not capable of prompt absorption is encapsulated. Therefore any foreign body placed in the peritoneal cavity to prevent adhesions most certainly induces that which it was intended to prevent.

All that the surgeon can do to prevent adhesions is to operate

with the utmost gentleness, placing the minimum of foreign bodies of all sorts, either temporary or permanent, within the abdominal cavity, and cover every denuded surface as carefully as though it were an unpardonable sin.

Bibliography

BAINBRIDGE: The Intra-abdominal Administration of Oxygen, Ann. Surg., 1909, xlix, 305.

BAUM: How to Avoid Adhesions in Abdominal Sections, Jour. Am. Med. Assn., 1894, xxiii, 242.

BLAKE: The Use of Sterile Oil to Prevent Intraperitoneal Adhesions, Surg., Gynec. and Obst., 1908, vi, 667.

BUSCH AND BIBERGEIL: Experimentelle Untersuchungen über Verhütung von peritonealen Adhäsionen, Arch. f. klin. Chir., 1908, lxxxvii, 99.

BYFORD: The Significance of Peritoneal Adhesions Following Operations, Surg., Gynec. and Obst., 1909, viii, 576.

CLARK: Anatomical Considerations in Peritoneal Adhesions, Surg., Gynec. and Obst., 1909, ix, 603.

CLEVELAND: The Prevention and Relief of Post-Operative Intestinal Obstruction, Med. Rec., 1901, lix, 1.

COFFEY: Abdominal Adhesions, Jour. Am. Med. Assn., 1913, lxi, 1952.

CRAIG AND ELLIS: An Experimental and Histological Study of Cargile Membrane, Ann. Surg., 1905, xli, 801.

CUBBINS AND ABT: A Preliminary Report Concerning the Effect of Foreign Substances in the Peritoneal Cavity, Surg., Gynec. and Obst., 1916, xxii, 571.

DEMBOWSKI: Ueber die Ursachen der peritonealen Adhäsionen nach chirurgischen Eingriffen, Arch. f. klin. Chir., 1888, xxxvii, 745.

GELLHORN: Experimental Studies of Post-Operative Peritoneal Adhesions, Surg., Gynec. and Obst., 1909, viii, 505.

v. HIPPEL: Zur Nach und Vorbehandlung bei Laparotomien, Zentralbl. f. Chir., 1907, xxxiv, 1345.

KELTERBORN: Versuche über die Entstehungsbedingungen peritonealen Adhäsionen nach Laparotomien, Zentralbl. f. Gynäk., 1891, xiv, 51.

KLATZ: Behandlung des Ileus post operationen, Zentralbl. f. Gynäk., 1892, xvi, 977.

LAUENSTEIN: Verwachsungen und Nelzsträuge im Leibe als Ursache andauernder, schwerer Koliken, Arch. f. klin. Chir., 1893, xlv, 121.

MALCOLM: On the Importance of the Practice of Washing out the Peritoneal Cavity as a Means of Securing a Natural Disposition of the Intestines after an Abdominal Section, Lancet, London, 1890, i, 75.

MARVEL: Prevention of Peritoneal Adhesions by Adrenal Salt Solution, Jour. Am. Med. Assn., 1907, xlix, 986.

MICHAEL: Zur Fibrolysinbehandlung perigastritischer Verwachsungen, Berl. klin. Wchnschr., 1907, xliv, 1606.

MORRIS: A Report on Experiments Made with Cargile Membrane for the Purpose of Determining Its Value in Preventing the Formation of Peritoneal Adhesions, Med. Rec., 1902, lxi, 773.

MÜLLER: Zur Nachbehandlung schwerer Laparotomien, Arch. f. Gynäk., 1886, xxviii, 448.

OFFERGELD: Zur Behandlung gynäkologischer Erkrankungen durch Thiosinamin, München. med. Wchnschr., 1905, lii, 1769.

PATON: A Clinical Lecture on Some Observations on Intra-abdominal Adhesions, with Illustrative Cases, Med. Press and Circular, 1907, lxxxiv, 30.

RICHARDSON: Studies on Peritoneal Adhesions, Ann. Surg., 1915, liv, 758.

RÜBSAMEN: Tödliche Kompfervergiftung nach Auwendung von offizinellen Kompferöl zur postoperatiren Peritonitisprophylaxe, Zentralbl. f. Gynäk., 1912, xxxvi, 1009.

SCHEPELMANN: Das öl in der Bauchchirurgie, Arch. f. klin. Chir., 1912, xcix, 879.

STAVELY: The Treatment of Post-operative Adhesions, Washington Med. Ann., 1910-11, ix, 1.

SZNBINSKI: Thyosiamin und Litrolysin in ihren Bezuhungen zu Gelenkerkrankungen, Charité Ann., 1909, xxxiii, 468.

TINKER: Prevention of Adhesions after Abdominal Operations, Am. Med., 1907, n. s. ii, 284.

UYENO: Ueber das Schicksal der peritonealen Adhäsionen und ihre Beeinflussung durch mechanische Massnahmen im Tierexperiment, Beitr. z. klin. Chir., 1909, lxv, 277.

VOGEL: Klinische und experimentelle Beiträge zur Frage der peritonealen Adhäsionen nach Laparotomien, Deutsch. Ztschr. f. Chir., 1902, lxiii, 296.

WEBSTER: The Prevention of Adhesions in Abdominal Surgery, Surg., Gynec. and Obst., 1909, viii, 574.

WILKIE: The Use of Oil in Abdominal Surgery, Surg., Gynec. and Obst., 1910, x, 126.

CHAPTER VIII

CHANGES IN THE CIRCULATION

Since the vessels empty after death, it is necessary, in order to gain a mental picture of the normal state of the circulation of the peritoneum, to observe a large number of abdomens in the living. In children there is a relative constancy in the fullness of the vessels, but in the adult there are so many individual variations, not always accounted for by the presence of any obvious pathologic process, that it is difficult to obtain a concept of the normal average. The variations in the vascular supply of the peritoneum are no more striking than are those of the skin, and they seem, in a general way, to be responsive to the same conditions and influences. It must be expected, therefore, that the degree of prominence of the vessels of the peritoneum will bear some relation to the general vascular habit of the individual.

As we view the peritoneum during the course of an operation on the intraabdominal organs we see vessels everywhere beneath the glistening sheen of the peritoneum. For the most part these are vessels destined for the supply of some parenchymatous organ and are covered only by the peritoneum. This relationship is most readily appreciated by dissecting off the peritoneum from such vessels. It is only near their termination that they give off branches which come in intimate relation with the peritoneum.

The peritoneum, because of its translucency, permits the color of the organs it covers to shine through it, as is well illustrated in the case of the liver and spleen. The peritoneum appears pearly over fascial surfaces and over parenchymatous organs when there is a thickening of their capsule, as is noted most frequently in the case of the spleen in perisplenitis. Over the intestine the peritoneum permits the coloring of unstriped muscle fibers to appear. In the case of the large intestine the color over the teniæ is pearly white because of the heavy bands of fibrous tissue in these structures. When the gut is much distended the gases of the intestine may give

the whole a nearly transparent appearance. It is obvious, therefore, that, when peculiarities are observed, it is necessary to determine whether the change is in the peritoneum itself or in the organ it covers.

In the mesentery the peritoneum is transparent between the vessels, except close to the vessel walls, where there is a deposition of adipose tissue. With the repeated division of the vessels, as the gut is approached, this perivascular fat increases, and the area of transparency narrows proportionately. The amount of fat deposited about the vessels is subject to the greatest individual variations. In obese persons the entire area between the mesenteric vessels may be obscured by a fat deposit. This is particularly true of the terminal loops.

After the vessels enter the gut they are visible as concentric rings. They are most prominent in the proximal portion of the gut and during the periods of functional activity. When the gut wall is contracted the vessels may be obscured so that only the diffuse color of the muscle fibers can be seen through the peritoneum.

The degree of distention of the vessels is easily influenced by irritation and changes in intravascular pressure. When respiration is interfered with in the course of the anesthesia it is often first manifest in an increased distention of the mesenteric vessels. An injury to any part of the abdomen is responded to by dilatation of the vessels over a wide area. The irritation from a gauze pack, temperature change, etc., quickly alters the prominence of the vessels. In fact the classic experiments conducted by Cohnheim on the ears of rabbits may be repeated on the peritoneum.

We may follow the analogy of the peritoneum and the skin in the study of the significance of the dilatation of the vessels. In certain regions of the skin the presence of smaller vessels is manifest only in a diffuse coloring, as the pinkness of the cheeks. In the skin, in response to certain stimuli or disturbances in vascular mechanics, fine vessels in profusion become visible, where before the surface presented a uniform appearance. Familiar examples may be noted in the skin of the nose of the alcoholic or in the face of the teamster or engineer. The dilated vessels of the teamster bespeak protection against the windy blasts, and of the engineer to the stimulus of heat. The dilated vessels in each case represent

compensatory processes. The nose of the tippler, an index of the joys of an epicurean life, represents the physiologic action of a drug, and it should not surprise us if the cecum, much nearer the source of his joy, should present similar evidence. The legs of multipara may bear evidence of a biological obligation freely met. None of these conditions can be considered pathologic, but, in response to the factors mentioned, the vessels become dilated and remain distended, and become permanently visible to the naked eye. It is only when these vessels become of a size to be attended by secondary processes which annoy the individual that they may be regarded as pathologic.

There is a corresponding absence of a sharp border line between the normal and the abnormal in the case of the peritoneum. Variations in the prominence of vessels correspond to certain general physical states and to certain age periods. In the invalid and in the aged, due to general sluggish circulation and to atrophy of the perivascular tissue, vessels become visible which were previously not so. This greater conspicuousness of the vessels is due not to change in the vessels, but in their environment; and with a restitution of the individual to his normal state the vascular condition cares for itself.

The peritoneum, like the skin, normally free from visible vessels, except in certain definite situations, presents, under certain general or special conditions, vessels visible to the naked eye. As already noted in the section on anatomy, there are in the transparent areas of the peritoneum, invisible channels which become visible under stress. These normally invisible channels evidently give passage normally to plasma, as the spaces in the cornea; and it is only in response to the stress of reactive processes that they become avenues of corpuscular migration. Like the vessels of the skin, when the causative factor remains permanent, or so long that definite vascular or perivascular changes have taken place, the vessels remain permanently visible. So in the skin, too, mere visibility does not present evidence, *pari passu*, that the state is pathologic. These facts may be stated without comment as regards the skin, but as applied to the peritoneum they are yet the subject of a lively controversy. Their causative relation to certain visceral symptoms can not perhaps be categorically denied merely because of the

morphologic similarity to certain lesions of the skin. On the other hand, it is worth remarking that their presence does not warrant an assumption of etiologic relationship to the symptoms the patient may be complaining of at the time of the observation.

In order to approach a satisfactory presentation of this problem it is necessary first to regard the facts in the light of general circulatory mechanics and the changes in other tissues incident to the changes in the circulation; and, secondly to compare the state of the peritoneum with the symptoms of which the patient may complain.

It is not always easy to state what shall be regarded as an abnormal emptiness or fullness of the vessels. Functional activity influences the amount of blood in an organ. Thus Ranke estimates that the amount of blood in a muscle when it passes from a state of rest to a state of tetanus, is almost doubled. Functional activity is never so great as to cause a dilatation of the potential vessels; but the service vessels during the height of digestion show an increased fullness yet they return to normal when the stress of work is removed. Likewise functional activity in the neighboring organs is never so great as to cause a filling of the potential capillaries. When vessels are visible where none can be seen normally there is congestion due to reactive process.

In order to approach the investigation of the circulatory changes of the peritoneum in a systematic manner it seems best to adopt the general classification followed in general pathology. To this classification must be added the special problems relating to questions of etiology and clinical significance which the peculiarities of this region make necessary. Active and passive hyperemia occur in the peritoneum according to general laws governing any vascular tissue. The passive hyperemia of the peritoneum is sometimes complicated by secondary changes peculiar to the peritoneum. The permanent dilatation of vessels not ordinarily functionating adds pictures not generally recognized.

1. Anemia.—The peritoneum, together with the subperitoneal tissues, becomes anemic when the general body suffers an impairment of nutrition. This is a hematologic, and not a topographic-anatomic problem, and it concerns local anatomicopathologic condition only in so far as it reduces the defensive forces of the region.

In general anemias the peculiar pale appearance of the abdominal organs is due more to the loss of hemoglobin than to any change in the diameter or fullness of the vessels of the peritoneum itself.

Actual narrowing of the lumen of the vessels of the peritoneum, the result of endovascular changes, may lead to anemia of the peritoneum. This follows endarteritis from old age or from syphilis. The narrowing may lead to functional impairment of the muscle coats and of the glands of the intestines, as well as of the peritoneum. Usually, the latter is not sufficient to produce changes of importance unless a thrombosis becomes associated with the narrowing. The great paleness of the peritoneum in the aged is but a part of a picture of the general bodily conditions, and has no special significance.

The estimation of the influence of vasomotor control over the vascular content presents many difficulties. Chilling the surface of the abdomen produces a temporary constriction of the peritoneal vessels. Splanchnic stimulation acts in a like manner. Whether this part of the body may suffer a vascular constriction from great emotion is difficult to say, but there are clinical facts that make such an assumption plausible. Every instructor has noted an increased demand for stomachics among students as the term examinations approach. Similarly a vasomotor neurosis, comparable to the constriction of the vessels of the hand in certain nervous states, may not be beyond reason.

When functional inactivity involves the underlying organ the overlying peritoneum suffers an associated change. Thus the peritoneum, covering areas of muscular paralysis, pales after the section of large nerves supplying the abdominal wall. The same is true of the peritoneum covering an organ, as over a splenic infarct when the underlying parenchyma has become functionless. Apparent thickening of the peritoneum in such situations is due to the formation of fibrous tissue of the capsule of the organ and in the subperitoneal tissues, while the peritoneum itself has suffered an impairment of its functional capacity because of a cicatrization about the subperitoneal vessels.

2. **Hyperemia.**—The reader, unless he is a sophomore medical student, will not be offended if he is reminded that by hyperemia is understood a state in which the amount of blood in an organ is

greater than normal. This increased amount of blood may occur in spite of a normal flow toward the organ when the flow away from it is slowed. On the other hand, hyperemia may be due to an increased flow of blood to the part with a normal or decreased outflow. What is still more important, a state of hyperemia exists so long as the inflow exceeds the outflow, even if the outflow exceeds the normal rate. The former type is called "passive," the latter "active" hyperemia.

These types are by no means so easily separated as this simple scheme seems to indicate. The reason is that a passive hyperemia may so interfere with nutrition that a reactive process, with an associated increased flow of blood, is set up, thus instituting processes similar to those which take place about an infarct. On the other hand, an active hyperemia in response to the injury, may exist for so long a time that permanent vascular changes in the neighborhood occur which would be only temporary were the lesion of shorter duration. The result is that, when the noxious influence ceases to act, the vessels remain dilated, and may continue as a menace to the general nutrition of the tissues. In organs, such as the peritoneum and omentum, where the tissues surrounding the vessels are so small in amount as to give them but scant support, the vessels recover with greater difficulty than in more solid organs. These factors make the problems in this region particularly complex.

1. *Active.*—Increased flow of blood to a given area of peritoneum occurs either in response to irritation from some underlying organ or to the burden of supplying, in whole or in part, an organ deprived of nutriment or to combat a local irritation.

The hyperemia due to functional activity of the organ can be neglected here. For instance, during the process of digestion the surface of the stomach becomes of a deeper color. The increase in color is due to a dilatation of the deeper vessels of the functioning layer of the stomach; and the modification of the color of the peritoneum shows in a measure in the influx of blood. More marked changes may be noted in the peritoneum covering the pregnant uterus, where there are structural changes corresponding to the changes in functional activity in the uterine wall itself. In this case hyperemia of function may remain when the extra demands of function cease. This hyperemia, the result of repeated preg-

nancies, may remain when the woman no longer conceives. Similar conditions may occur in some other regions. This is often marked

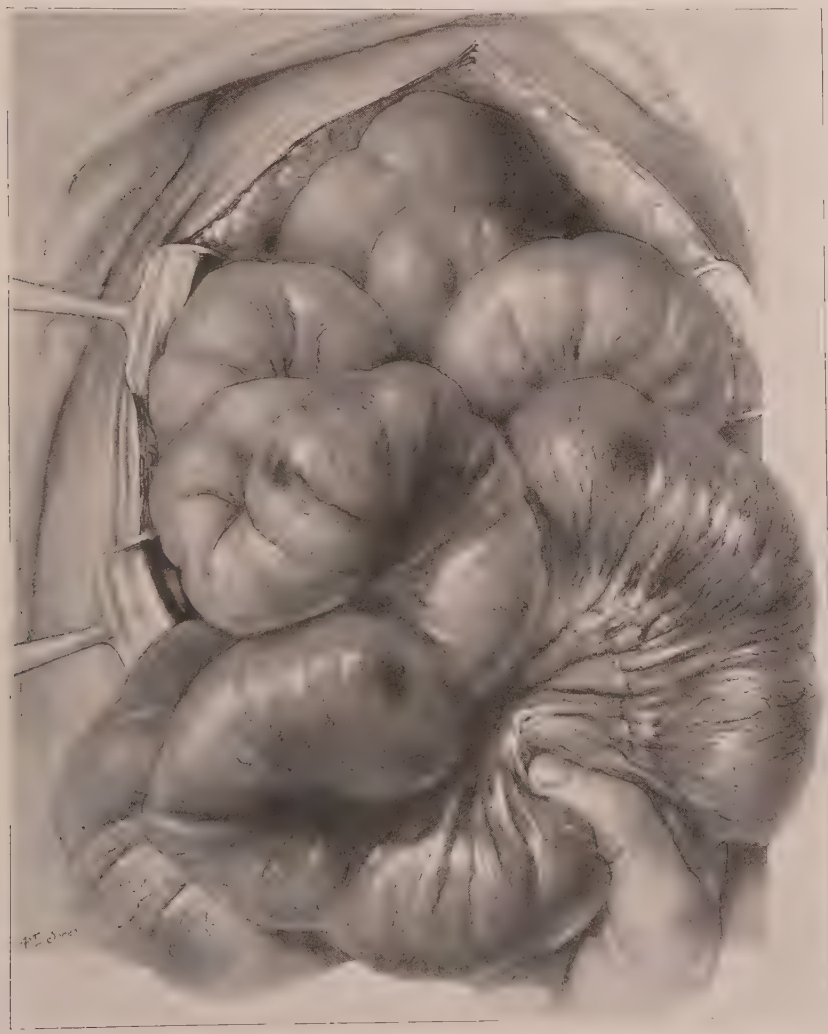

Fig. 131.—Dilatation of the vessels of the mesentery and intestinal wall in a case of intestinal obstruction due to carcinoma of the cecum.

by an extensive plexus of vessels in the peritoneum covering the broad ligament. (See Hertzler: Pericolic Membrane of the Broad Ligament, Surg., Gynec. and Obst., 1913, xiii, 60.)

The hyperemia due to irritation can best be studied in lesions in which the peritoneum is not the site of an inflammatory exudate. In intestinal obstruction irritation from retained products causes dilatation of the vessels of the peritoneum covering the obstructed

Fig. 132.—Dilatation of the mesenteric vessels caused by the presence of an aseptic pledget of gauze. The omental vessels not in direct contact with the foreign body also show marked dilatation.

gut (Fig. 131). The same principle can be more strikingly demonstrated by experiment (Fig. 132). By placing a pledget of gauze in the abdomen there is an immediate hyperemia, not only about the foreign body (Fig. 133), but throughout the abdominal cavity. In the illustration the dilatation of the vessels in the abdominal

wall and the omentum at a distance from the chief point of irritation are even more pronouncedly dilated than are those immediately surrounding the gauze pledget. This experiment illustrates very well how the site of ultimate hyperemia may be far removed from the site of the original infection.

A local lesion may produce a hyperemia in its immediate vicinity, but extending some distance from the point where there is active inflammatory reaction. For instance, about an incision through the peritoneum in which the healing is purposely delayed, a wide field shows dilatation of the vessels (Fig. 134). If long enough con-

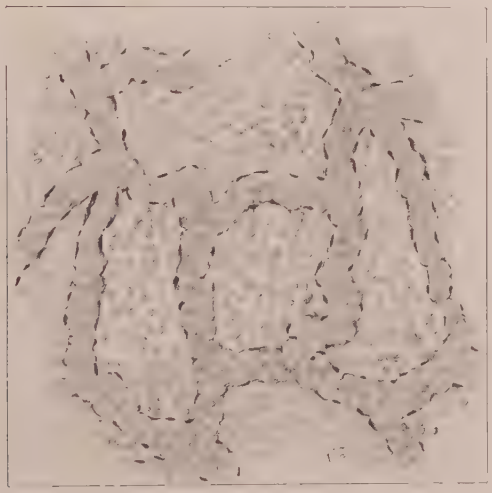

Fig. 133.—Microscopic reproduction of an acute hyperemia. The area here presented represents a "bloodless" field in the mesentery.

tinued, the venous dilatation becomes permanent though the vessel walls have never suffered reaction. The mechanical stress resulting from prolonged dilatation must be the determining factor.

Usually the hyperemia, due to inflammation of the underlying tissue, subsides when that tissue recovers. This is less likely to occur the longer the underlying organ is diseased. In the later stages of such associated congestion both processes become so involved that it is only by studying the genesis of the various factors that a degree of clearness of comprehension can be obtained relative to the etiology. That is to say a chronic affection of the

mucosa may cause a dilatation of the peritoneal vessels which becomes permanent without being a matter of clinical significance, after the original lesion is recovered from. For instance a congested cecum in association with a chronic appendicitis may cause a dilatation of the vessels of the peritoneum which does not disappear when the cecum, as a result of the removal of the appendix, has returned to normal. Often, too, about an old gastric ulcer a permanent dilatation of the vessels of the peritoneum may remain long after the ulcer has healed. (Compare Fig. 118, in the chapter on adhesions.) In each of these instances a permanent dilatation

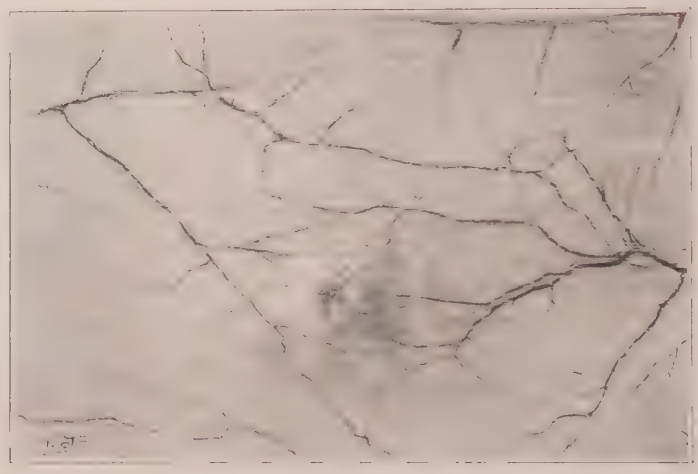

Fig. 134.—Dilatation of the vessels of the parietal peritoneum about the site of an old aseptic drain opening.

has taken place as the result of an active hyperemia long maintained, but which has ultimately subsided. In such instances the cause of the dilated vessels must be sought in healed lesions. This relationship between present and past lesions has been too often overlooked by surgeons in interpreting changes in the circulation of the peritoneum.

In a second type of active hyperemia of the peritoneal vessels the relation is more clearly seen; if, for instance, after an accident to an organ or structure has occurred, irritation of the peritoneum of neighboring organs develops causing them to adhere to and supply it with nutrition. This is best observed in myomas of the uterus or

in ovarian cysts when the usual channels of nutrition have become insufficient because of the thrombosed vessels or twisted pedicle. The irritation of tissue threatened with necrosis is sufficient to cause an attachment of the adjoining peritoneum and a dilatation of the surrounding vessels. The most pronounced conditions are seen when the omentum becomes attached to a myomatous uterus. In such instances enormous dilatation of vessels is often noted. Inasmuch as the primary dilatation of the vessels occurs as a response to stimulation from dying tissue, the hyperemia may be regarded as active, though the most conspicuous increase in size is manifested by the veins. The purpose of such venous dilatation is to carry off the excess blood brought by the new attachments. Because of their thin walls this sudden excess of contents causes them to become mechanically dilated. This mechanical distention is gradually added to the natural dilatation which occurs in response to injury, and increases after the primary stimulation has passed. This dilatation adds a passive factor to the already complicated problem surrounding the active hyperemia. In this way the passive aspects may become dominant, or, more correctly stated, may overshadow the more important active hyperemia. In such instances, if the demand for the hyperemia ceases, a state corresponding to varicosities in other parts of the body, is reached. This will be discussed under a separate heading.

In such cases it is only by reading backward from the evidence at hand that clarity can be obtained. For instance, evidence of a twist in the pedicle of an ovarian cyst may account for the dilatation of the vessels of the anterior parietal peritoneum. The important point to the surgeon is that, when such a state is noted, it is evident that there is, or was, a disease process somewhere in that region. A correct solution is then dependent on his ability to read the message which remaining lesions or scars convey.

From the foregoing it is evident that the relation between active and passive hyperemia, once the disease has passed the active stage, is a complicated one. The dominant lesion, as a matter of fact, is the passive venous hyperemia, usually static in character. For this reason the chief discussion of the structural changes associated with vascular dilatation is reserved for the chapter on varicosities.

2. *Passive.*—The discussion of passive hyperemia conventional to

general pathology is inadequate for the study of the circulatory changes in the peritoneum. The reason for this is that the vessels, being thin walled and unsupported, respond more readily to static and reactive influences, and, for the same reason, recover their normal state with greater difficulty. As in other regions of the body, passive hyperemia is caused whenever there is a mechanical retardation of the venous flow. This may be due to some pathologic process within the abdomen or to some factor in the liver, lungs or heart. The most prominent examples of hyperemia of the peritoneal vessels are seen in adhesive pericarditis. In most instances passive hyperemia of the peritoneum is caused by pathologic processes within the hollow viscera which are not severe enough to cause a reactive stimulus to the circulation. The veins of the peritoneum dilate with those of the organ, and, because of the conditions above indicated, show the effect of the lesion long after the primary disease has ceased. Differentiation of a primary passive hyperemia from one secondary to an acute process is not easy. A distinction may be made in many instances by noting whether the dilatation only of vessels usually visible has taken place, or whether there is the appearance of vessels where none are normally seen; that is, whether the potential vessels have become active. When the latter fact obtains, it may be assumed that the peritoneal vessels have responded to an increase in the larger, more deeply lying vessels because of some acute inflammatory process. In addition to the causes above cited a passive hyperemia is often seen associated with splanchnoptosis. Dilatation of the vessels of the broad ligament coincident with uterine displacement, is a common occurrence. They resemble passive dilatation of the superficial vessels of the leg, with which they are not infrequently associated.

Sometimes passive hyperemia is the result of a limited lesion above, resulting from a past disease such as a healed cholecystitis or a gastric ulcer in which a vein has become narrowed in the process of cicatrization. In these passive hyperemias associated with obstruction above, the relation may be obvious when there are extensive adhesions about definite old scars; but in many instances, particularly when the peritoneum is viewed only through a laparotomy wound, this relationship may be overlooked. The lesions

about the stomach and gall bladder may escape detection, even if pronounced, when the lower abdomen, exposed through a lower abdominal incision, is brought into view. Even an upper abdominal incision may require close analysis before the relation can be established. Even at the autopsy all points can not be followed out to a satisfactory degree because of the emptying of the vessels when the circulation ceases.

Varicosity of the Peritoneum.—A varicosity may be understood to be a condition of permanent dilatation of the veins of a part, usually associated with secondary changes in the structure of the vessel wall and in the surrounding tissues. It is due to a long continued irritation in the neighborhood of the vessels or to some condition which obstructs the venous flow for a period of time long enough to permanently injure the elasticity of the vessel wall. This definition is as applicable to the condition when found in the peritoneum as when found in the skin. (Compare Hertzler: Pseudoperitoneum, Varicosity of the Peritoneum and Sclerosis of the Mesentery, Jour. Am. Med. Assn., 1910, liv, 351.)

The direct relation of the change in the peritoneum to other varicosities may be studied when the pampiniform plexus of the broad ligament and varicosities of the legs are associated in the same patient. (See Hertzler: Pericolic Membrane of the Broad Ligament, Surg., Gynec. and Obst., 1913, xiii, 60.)

It is noted in the discussion of acute hyperemia that the condition frequently terminated in passive state. When this is reached, dilated vessels remain as landmarks of past lesions. The changes which determine a permanent dilatation of the vessels may subside and the vessels themselves, the result of some other condition, may produce, in turn, other lesions. They become in such instances both the result and cause of disease.

In general, the two types of hyperemia, the passive and the active, find expression in two types of varicosities, the static and the reactive. The separation of the two conditions is of importance clinically, for, the one being an expression of constitutional defect, is never a surgical problem; while the other, being the end result of a local disease, may, like varicosities in other situations, at times present problems requiring relief by surgical means.

The Static Type.—In this type there is merely an exaggeration

in size of the vessels normally visible to the naked eye. Some vessels previously but faintly visible may stand out prominently. Among these may be noted the terminal vessels of the arterial intestini attenuis and their ramifications between the muscular layers of the gut and between the muscle and the peritoneum. Vessels in the folds of the peritoneum, normally but slightly conspicuous, particularly those of the mesoappendix, the "bloodless" folds of Treves, the paracolic ligament, and analogous associated bands between the colon and parietes, are the ones most likely to strike the eye. The same thing applies in a lesser degree to the other peritoneal-covered ligaments. The omental and mesenteric vessels of regular distribution also stand out as prominent blue cords.

Associated with this type is usually a general visceroptosis. The patient is usually tall and long waisted. The organs hang loose within the abdomen. The abdominal wall furnishes little support to the abdominal contents. There is a general laxity of the entire bodily musculature, and the general mental and nervous condition is quite as lax as the muscles. There is usually an underlying tendency to relaxation, hereditary to the individual, but often it is acquired by hard work, child-bearing and marital excesses. This is often augmented by intercurrent acute diseases, general and pelvic.

The opposite type may be represented. In plethoric persons the peritoneal vessels are as often surcharged as are those of the skin. In such persons the entire circulatory apparatus is full, but the prominence of the vessels is likely to be inconspicuous because of the abundance of perivascular fat.

The personal equation of the widest possible margin must be allowed the observer in the study of this condition. Vessels always visible are more prominent than in the average patient. Whether this has always been so, that is, whether it is a congenital variation, or is the result of some of the factors above noted, must be determined by the judgment of the observer. In any abdomen vessels stand out more prominently in some regions than in others; and in judging the relative vascular fullness, the general state of the patient must be carefully noted.

Analogy would compel the question whether or not there may be

sequelæ, either in the gut or in the peritoneum, analogous to eczema of the calf or varicose ulcers, which so frequently complicate long-standing varicosities of the leg. Here, as is well known, as a result of a simple stasis, an ulcer may form which may require surgical interference. No analogous organic lesion has been demonstrated in the peritoneum or the subjacent gut. Functional disturbances, notably constipation and diarrhea, have been ascribed to such a condition, but proof of a direct relationship has been lacking. Possibly the dilated vessels may provide and enhance such conditions. Excessive secretions, finding expression in diarrhea, may well bear some relation to the hyperemia. Constipation, functional in character, may be aggravated by malnutrition of the muscle coats resulting from ill nourishment of the intestinal musculature. These questions can find no answer in clinical observation; and postmortem study offers little aid, for after death the vessels empty and thus there will be little evidence of the previous condition.

Objection is here entered against ascribing to the condition under consideration a pathologic significance unwarranted by available facts. The dictum of my cult is all too universal: that which is not understood should be removed. In no lesion has this tendency been more prominently exhibited recently than in vascular peculiarities of the abdomen.

The anatomic changes in the purely static type of varicosities are simple. The vessel walls usually exhibit a simple atrophy, but the media may be the site of a fibrosis. There is little or no change in the perivascular tissue; therefore the vessels stand out as blue cords. The vessel walls exhibit little change indicative of an attempt to produce added strength in order to sustain the heavier column of blood.

In the plethoric individual the vessels show no change in structure. The general tonus of such a person has not suffered, and the vessel walls probably receive support from the more abundant perivascular adipose tissue. A person may be, of course, both fat and flabby. In such a case huge vessels may be noted. Operations for umbilical hernias give frequent opportunities for the study of this condition in such persons.

Reactive Type.—Vastly more striking than the static type just described are those in which there is a permanent vascular dilata-

tion, the result of some predisposing disease. In many regions the lesion responsible for the vascular dilatation is still in evidence (Fig. 133). The general relation of such previously existing processes, which cause the vessels to become permanently dilated, is due to the changes that have taken place in the walls of the vessels and in the surrounding tissue while the vessels were dilated in response to injury. A detailed study of these changes is now in order.

Varicosities representing end processes are found most often in those regions where inflammatory reactions take place the most frequently, namely, in the ileocecal and hepatic regions. This fact alone suggests a relationship to the reactive processes. The gross appearance is often very striking. The vessels normally visible are present in an exaggerated degree, though sometimes the reverse is the case. The most striking feature is the fine plexus of vessels, the main ones of which run parallel with the main subperitoneal fibrous bundles, but which are united by smaller transverse vessels. Howard Hill has suggested that it appeared as if a coarse comb had been drawn over the peritoneum, each tooth of which left in its wake a new vessel. The result is that, where previously there were no vessels visible to the naked eye, there now appears an extensive vascular network. Their source can be determined from a study of the structure of the vascular system in the peritoneum. As already discussed in the chapter on histology, there are in the apparently avascular areas of the peritoneum channels permeable for silver solution, which do not transmit red corpuscles under ordinary conditions. These vessels may be seen to take up a part of the burden of the circulation in times of stress; and, occupying the anatomic direction of the new vessels above mentioned, it seems fair to assume that the origin of the parallel vessels in varicosities lies in the vessels ordinarily inactive, which I have called the potential vessels as discussed in the chapter on physiology.

The main direction of these vessels is that in which the organs travel during the period of development to reach their permanent location. In the case of the cecum the veins are usually directed from the parietal wall obliquely downward and inward; or in cases in which the cecum descends before it becomes attached they extend from without inward in a line with the transverse axis of the

body. The explanation of this direction is found in the fact that the vessels are developed early before the final position of the cecum is attained. As the cecum descends, their direction changes from directly inward to the common direction of downward and inward. If, however, the cecum does not become attached until it attains its final position the direction remains directly inward above, and inward and upward below the ileocecal valve. It is possible, therefore, from the direction of these vessels to judge how far the cecum migrated after it became attached.

The same embryologic facts may be employed in predicting the direction of these vessels in other regions. When the vessels are prominent in the cecum and its mesentery, the vessels invariably point to the root of the mesentery, no matter where the cecum lies. In the region of the gall bladder they run parallel with the main fibers of the hepatoduodenal and hepatocolic or other ligaments of this region. In the region of the sigmoid they are parallel with the main fibers of the original sigmoid mesentery. Owing to the more certain steps the organs in this last-named region take in their development, there is less variation in the change of direction in these than in the vessels of the cecal region.

The vessels themselves present a varying histologic picture, depending upon the method employed in their study. If they are fixed by the injection of a solution into their lumen they remain as relatively thin-walled vessels. When hardened in formalin or alcohol, particularly the latter, they are contracted and relatively thick walled. The thickness varies in different specimens, apparently depending upon the character and duration of the reactive process that was responsible for their origin. In this they present the same characteristics as varicosities elsewhere. Those who have studied varicose vessels from all regions, I dare say, have been astonished to find vessels which appear as large as a finger in the operating room appear as thick-walled vessels with small lumen when seen under the microscope.

The characteristic histologic change in the vessel wall is usually a hyperplasia and with hyaline degeneration of the media (Fig. 135). There may remain evidence of reactive activity in the intima manifest by the presence of several layers of endothelial cells (Fig. 136). This may be true when there is no evidence of a recent re-

active process elsewhere (Fig. 137). In some specimens it is evident that there has been a displacement of the muscular layer of fibrous tissue previous to the degeneration. The degree of change is very variable, and resembles in character the familiar change in

Fig. 135.—Permanently dilated vessels of the head of the cecum in a case in which the appendix had been repeatedly inflamed.

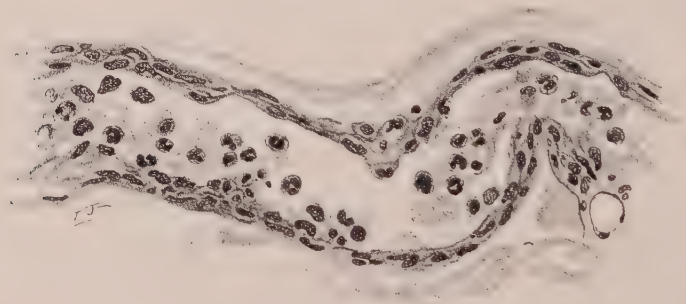

Fig. 136.—A vessel from a specimen similar to that presented in Fig. 135, but in a more acute stage. The vessel endothelium is several layers thick and there are a number of leucocytes adherent to the vessel wall.

vessel walls in uteri which have undergone involution, though there is not the associated reduction of the lumen.

In addition there is a subserous exudate immediately around the vessels which in some instances extends into the walls of the gut beneath (Fig. 138). This exudate imparts an increased mobility to the peritoneum, which makes it possible for the cecum to slip

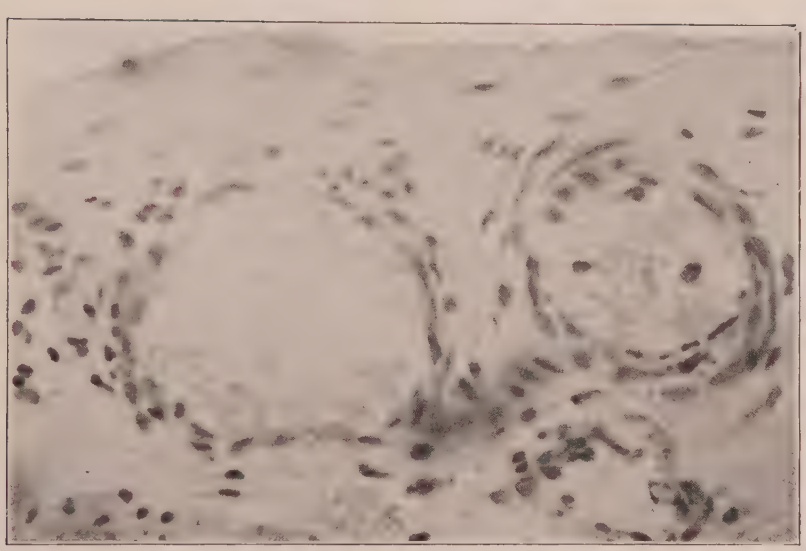

Fig. 137.—Vessel in which there still remains a hyperplasia of the endothelium after the reaction in the surrounding tissue has subsided.

Fig. 138.—Dilated vessels in the tip of omentum about a subacutely inflamed appendix in the fourth week. The organ is still much thickened by a hyaloid exudate in the connective-tissue network. This omentum was as thick as a hand.

about beneath the peritoneum "like a fetus in its membrane," as Frank Hall (quoted by Jackson) expressed it. This appearance led Jackson to designate it the pericolic membrane.

The exudate seems to be colloidal in character, and resembles the exudate about varicosities in the legs. It varies in amount, from barely sufficient to be identified microscopically, in some cases, to thick masses easily recognizable by the naked eye, in others. When abundant, as I have seen it adjacent to a subacutely inflamed appendix, it resembles the edema produced when an anesthetic solution is injected beneath the peritoneum.

The subperitoneal tissue is usually affected to a considerable degree. The connective tissue usually retains its specific affinity for dyes, but, when more extensively affected it may lose its specific reaction to Van Gieson's stain, while retaining it unchanged for Mallory's stain. This is true particularly early when the inflammatory state giving rise to it is still in the subacute stage, say six to twelve weeks after an attack of acute appendicitis.

The underlying organs seem to suffer but little. The muscle of the gut wall may be infiltrated, but this takes place only in exceptional cases. A number of clinicians have repeatedly stated that the muscular coats of the bowels were thinned, but there seem to have been no microscopic studies made to substantiate the assertion.

The factors underlying this type of varicosities may be arrived at by two general lines of study. Known acute processes may be followed step by step. Of course, in the human this is not possible in a single patient, but by observing a large series the general trend can be charted with a high degree of probability. The other line is the study of fully developed cases. Signs of a past conflict may be found with the condition under discussion in many instances. Old appendiceal lesions, evidences of past cholecystitic processes or of an old gastric or duodenal ulcer with perigastric adhesions, are associated with permanent changes in the vessels now under discussion. The same holds true, it may be said parenthetically, of the vessels about any healed ulcer in any part of the body.

Unfortunately, these changes are often not so obvious as to impose themselves upon the notice of the surgeon untrained in pathology. The argument advanced against any assumption is that the area in which the dilated vessels are found is at some distance from the site of actual disease or in places where there is no evidence of a previous disease. When dilated vessels are found about

the cecum it is argued that the appendix can not be at fault because it appears normal while the condition now under discussion lies over the cecum and ascending colon. Yet when the appendices are subjected to microscopic study many of them show infiltration of the muscular coats with plasma cells. Often an increase of the subperitoneal connective tissue or a change in its tinctorial reaction, and an increase of the germinal cells in the follicles and not rarely a partial or complete obliteration of its lumen may be observed. These are evidence of a past inflammation though there may be no macroscopic evidence of disease.

A factor not sufficiently noted is that the milder inflammatory processes are the ones most apt to be followed by permanent dilatation of the vessels. The more virulent processes produce more temporary results. These statements can be readily substantiated by animal experimentation. In the human subject the same thing may be observed. In very virulent cases of peritonitis with much exudate there is much edema, leucocyte infiltration, and exudation. This exudate forms a granular fibrin which is subsequently absorbed, leaving the peritoneum and subperitoneal tissue as before. This sequence can often be followed in drainage operations for appendicitis. At the time of operation there are many adhesions, but later, when the secondary operation is done all these adhesions have disappeared. In the minor degrees of reaction the exudate forms a fibrinous exudate, which organizes into a permanent tissue, producing a permanent dilatation of the vessels. Each of these factors may come into play in the same case. Complete restoration may take place nearer the seat of reaction, while at a distance when inflammatory reaction was less intense the changes in the vessel wall may be permanent.

There is another reason why the appendix itself and the extreme end of the cecum should show the least vascular changes. The tip of the cecum is poor in blood vessels, as is the appendix, and when all the vessels in the quadrant are dilated this region suffers by contrast; furthermore, the vessels in this region may have suffered actual obliteration from a past process as is seen in the small atrophied appendices. Therefore, when the problem is examined closely, the very fact that the appendix is bloodless is the strongest argument that this is the region at fault in initiating the process.

The question of the state of the lymphatics is difficult to determine. Hall (quoted by Jackson) noted a dilatation of these vessels. It has been stated that the parietocolic peritoneum is devoid of lymphatics. This is not true, for they can be demonstrated in that situation by the aid of silver-injection methods, though they are not as numerous as in the mesentery (Fig. 139). Why lymphatics should dilate may be susceptible of several answers. They may be overfilled from their attempt to carry off the excessive exudate from the

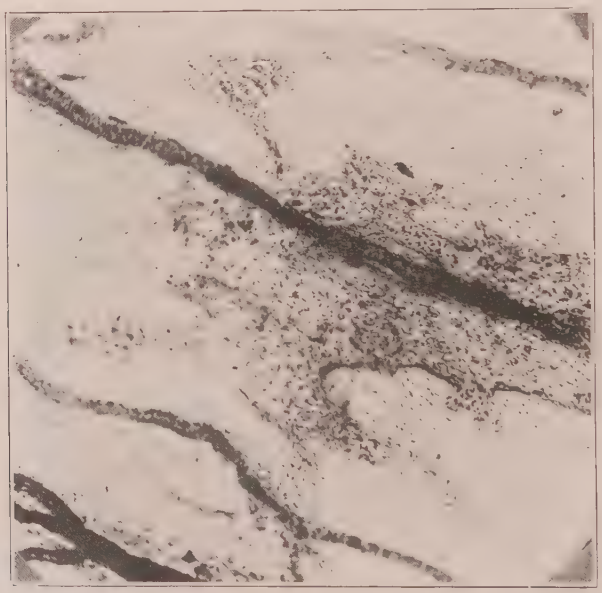

Fig. 139.—Dilated lymph vessels of the mesentery in a pericolic membrane. The straight channels are blood vessels, the wide, irregular lake-like channels are the lymphatics.

overloaded static veins. On the other hand they may be directly involved because of the occlusion of their own lumen by cicatricial contraction. This has been demonstrated in the region of ulcers. A great variety of other lesions in the right upper abdomen is capable of producing an occlusion of the lymphatics.

Opie reports experiments that are particularly apropos to the present discussion. He produced liver and gall bladder edema by means of cantharides. The edema was, at least in part, he concludes, due to thrombosis of the lymphatics; and there was a close

relationship between the thrombosis of the lymphatics and the degeneration of the cells. After ligation of the large lymphatic channels he was able to secure thrombosis by injecting bacteria into the blood, or by merely infecting the tissues in their neighborhood. The occlusion of the lymphatics so produced was followed by a transient edema. It is further interesting to note that in some of these animals a continued diarrhea followed occlusion of the thoracic duct.

In the light of these experiments the possibility must be admitted that a lymph-occlusion due to the inflammatory process might well give rise to a subperitoneal edema and to a vascular dilatation as well. In such cases it might be exceedingly difficult to demonstrate the lesion that gave rise to the primary occlusion or infection.

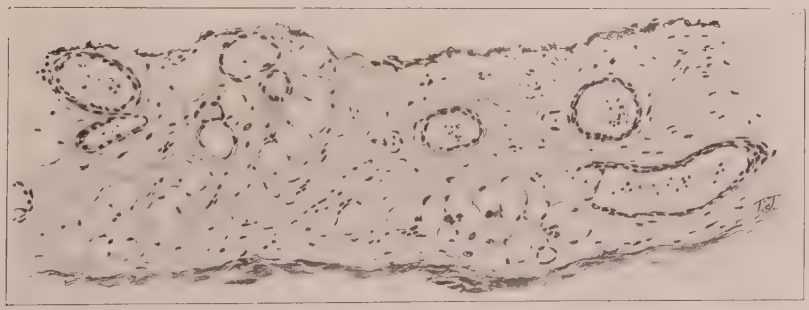

Fig. 140.—Selection of a typical pericolic membrane. Above is the peritoneum followed by colloid-filled tissue containing dilated vessels. Below is a layer of fibrous tissue often regarded as a newly formed peritoneum because of the colloid-thickened peritoneum above it.

The Pericolic Membrane. Such an extensive literature has sprung up on this condition under the euphonious title of "pericolic membrane," that a brief mention of the more important papers may serve a historic purpose.

So far as the literature on the pathology and pathogenesis of this condition is concerned, it is conspicuous only for its meagerness. In my own papers an attempt was made to emphasize the essential difference between varicosities, adhesions and pseudoperitoneums (Fig. 141). Frank Hall examined a specimen removed by Jackson and his account is the only other discussion of the pathology to be found (Fig. 140).

The paucity of literature on the pathologic study of this condition

Fig. 141.—A typical unusually well-marked pericolic membrane. The veil-like membrane is lifted from its usual position by the glossy subperitoneal exudate. The small vessels arranged parallel to each other are the outstanding feature. These vessels represent the potential vessels.

is in marked contrast to the colossal amount of literature which has sprung up in an attempt to make of this condition a *casus belli* in the belly of the patient.

Binnie was the first to observe peculiarities of the peritoneum in the cecal region, but he seems to have attached little significance to

it. Jackson in his inimitable manner presented it to the profession in a series of papers. After this time a large literature sprung up. It is cheering to note that Jackson himself was one of the first to conclude that the clinical importance of the condition he so graphically described had been very greatly overestimated; and he has lost no occasion so to state. Flint expressed the belief that the source of the membrane must be sought in the embryonal period. Flint is correct in believing that details in fetal development are responsible for the topography, but to this must be added a postembryonal irritant, which brings them into prominence. Eastman also speaks of them as representing fetal folds. He summarizes the various factors that may be active in different cases. Gerster advances the theory that chronic colitis exerts an influence on the production of this condition. This author speaks of the pericolic membrane as a pseudoperitoneum, which of course it is not. Finally, Eastman brings in the existence of chronic constipation in dogs as evidence in support of this source as a causative factor. It is certainly a far cry when we come to argue from the normal state of the dog to the quasipathologic conditions in the human subject. Eastman also discussed the condition as relates to the region of the sigmoid.

Bibliography

Binnie: Pericolitis Dextra, Month. Cycl. Pract. Med., 1895, viii, 341.

Eastman: Fetal Peritoneal Folds, Jour. Am. Med. Assn., 1913, lxi, 642.

Pericolitis Sinistra, Ann. Surg., 1914, lix, 41.

A Further Study of Pericolic Membranes, Surg., Gynec., and Obst., 1914, xviii, 228.

Flint: Embryonic Bands and Membranes about the Cecum, Bull. Johns Hopkins Hosp., 1912, xxiii, 302-311.

Gerster: On Chronic Colitis and Pericolitis, Ann. Surg., 1911, liv, 325.

Jackson: Membranous Pericolitis, Tr. West. Surg. Assn., 1908, xviii, 269.

Membranous Pericolitis and Allied Conditions of the Ileocecal Region, Ann. Surg., 1913, xlii, 374.

Opie: Lymph Formation and Edema of the Liver with Experimental Nephritis Produced by Cantharidin, Jour. Exper. Med., 1912, xvi, 831.

Thrombosis and Occlusion of Lymphatics, Tr. Assn. Am. Phys., 1913, xxvii, 449.

CHAPTER IX

INFLAMMATORY REACTION OF THE PERITONEUM

The bane of human life, as well as the preserver of it, is the reactive processes. Were it not for the capacity of tissue to set forth a series of phenomena calculated to combat the injury and to repair the defect, the securities of life of the infant animal would be rivaled by that of the lilies of the field. It is the capacity of the body to institute processes of self defense that give life security. Were it not that the animal is compelled to give heed to the presence of dangers that institute these reactions, injuries too great to admit of repair would be inflicted. The unpleasant sensations occasioned by injury and by the reactive processes that follow are the factors that impel us to lend our endeavors in immunizing the traumas and to aid the restorative processes in their effort to repair the mischief. These two phenomena, reaction on the part of the tissues, and the pain occasioned, are the fundamental factors in the subjective and objective study of the diseases of the peritoneum.

Until the advent of bacterial and serum diagnostic methods, the recognition of almost any disease was dependent very largely upon the demonstration of some form of inflammatory reaction or the changes which were the result of it, and the disturbances in sensation occasioned. In case of the peritoneum the discussion of its diseases is still a recital of the phenomena of the reactive processes which take place in it and the sensation which the changes produce.

The peritoneum owes its prominence in literature almost exclusively to the fact that it is so frequently the site of inflammation or can readily be made so to react. This prominence is due in part to the fact that the peritoneum itself is so often the site of disease but more particularly because it has been the most convenient object upon which to study the abstract problems of the reactive processes. The literature may, therefore, well be grouped under these general headings. Like all literatures, that dealing with facts preserves a permanent value while that dealing with the development of an

abstract theory may be devoid of enduring importance. So here those papers which record observations upon the diseases of the peritoneum retain an interest while much that related to the discussion of the nature of inflammation has lost its meaning. For instance, Hunter's work has a greater value than a large portion of the discussions or discoveries of the last decade.

The literature of the theoretic phase of inflammation is well preserved by von Recklinghausen, Marchand and to a lesser extent by Adami.

Only so much of the researches on inflammation, made on the peritoneum, as pertains to the diseased processes of the organ itself, need be considered here. These researches consist largely of the studies of Cohnheim, his coworkers and contemporaries. They embrace chiefly the studies of the phenomena occurring in the vessels and the relation of these to the extravasation of cells and their relation to the perivascular tissue. In this study along with these phenomena is considered the nature of the serous exudates which infiltrate the tissue and cover the peritoneal surfaces. This phase of the subject has not been hitherto adequately considered.

The problems which demand consideration are the following: If the simplest possible injury takes place without infection and without loss of tissue, healing follows along the simple lines noted in discussing the formation of connective tissue. If some of the tissue is destroyed the debris must be removed before the reparative processes can become operative. The activities by means of which dead particles are carried off are discussed under the rather abstract conception of phagocytosis. If bacteria enter the wound the defense functions of the tissue must become active. The bacteria must be destroyed and the toxins neutralized. After the defensive functions have been performed, then the part is ready to repair the defect produced by the injury and replace the tissue destroyed by it. If there has been a loss of substance this must be replaced by the production of new tissue. This last-named problem has been discussed in the section on wound healing by second intention.

Many pathologists, following the example of their forbears, make no dividing line between simple healing and the early stages of inflammation. The surgeon must ever recognize this division, narrow though the line may be. To him simple wound healing means healing

without infection, healing with inflammation implies infection. It is the maintenance of that narrow line between infection and non-infection that enlists his entire aseptic endeavors. Therefore, wound healing with its corollary, the formation of adhesions, as it occurs in clean wounds has been treated as an entity. The reactions instituted by injury must be treated as the antithesis of this, though separated often by an almost imperceptible boundary.

Etiology.—We may recognize at the outset that the processes which may set up a peritonitis may be so mild as to cause scarcely a disturbance in the general nutritional balance and can not be classed as inflammatory, if we insist on the presence of the classical signs of heat, swelling, pain and redness as emphasized by the older pathologists. We know from microscopic study that these phenomena may be present in all their elements long before they can be detected without such aid. A mere quantitative increase of these changes results in the definite gross signs of inflammation. The mild stimuli which excite the lesser reactions are as much to be regarded as causes of inflammation as the more intense degrees of action of the same stimuli. These milder manifestations have a great advantage as objects of study because they present the very earliest stages of the process. Many stimuli showing distinctive characteristics in their more intense forms are much alike in their milder manifestations. This fact is particularly pertinent in the study of the intricate changes in the peritoneum.

The peritoneum is vulnerable to all physical agents that will injure any other tissue. Exposure to air, to moist or dry heat, as well as the direct severing of its continuity may produce lesions of concern to the surgeon. The employment of foreign bodies or chemicals in the abdominal cavity whether used as remedial agents or employed to subserve some mechanical purpose, all are capable of producing lesions which must be repaired.

The degree of reaction excited is dependent upon the degree of destruction of tissue, but factors other than mere destruction of tissue enter. Carbolic acid may destroy tissue without inciting to reaction, while iodine incites to reaction without destruction of tissue. Bacteria are of course the prime causes of inflammation of the peritoneum. These like many purely chemical factors produce reaction in very varying degrees.

The vulnerability of the peritoneum to the several agencies to which it may be exposed is so variable that these may be studied separately.

Reaction Due to Drying.—The peritoneum, being provided with a fluid covering which acts as a lubricant, endures exposure badly because of the readiness with which this evaporates. Within a few minutes after the peritoneum is exposed to the air the surface begins to lose its luster. The fluid condenses and remains as a delicate pellicle over the endothelial cells. During the first 15 to 30 minutes the cell itself is not affected and if the normal environment is restored, the fluid normally covering it reaccumulates and normal conditions are restored. If the exposure is continued from 30 to 60 minutes, the cell itself becomes dried. The protoplasm becomes condensed and though the lessening in volume is not very noticeable the total shrinking amounts to not less than a fourth of the thickness of the cell. With even more complete drying the edges of the cells retract and no longer form a complete covering for the basement membrane. If the drying process is allowed to advance still farther, the basement membrane suffers. When this occurs the cell is loosed from its base. After the drying process has extended to such a degree the cell is not restored to its former state, but a series of reactive processes must be instituted before replacement of the cells can take place. This is marked by a dilatation of the unaffected vessels which results in an increased supply of blood in the region. During the time of exposure, however, there is no visible increase in the size of the vessels nor is there exudation. If an exudate forms, it is prevented mechanically by the dried structures above from reaching the surface. It is only after the membrane is returned to the abdomen that the vessels adjacent dilate and an exudate begins to form. As the fluid accumulates beneath the cells they are lifted from the limiting membrane and must be replaced by new cells. If drying continues far enough, the vessels become occluded and death of the tissue follows. Such extreme degrees of desiccation are noted only in animal experimentation.

The drying to which the peritoneum is exposed in the operating room does not affect the cells in a deleterious manner. At most the surface loses its glistening appearance. As soon as the peritoneum is returned to its normal surroundings the fluid is quickly

reformed. The vessels undergo a limited degree of dilatation, but in the course of a few hours the circulation is restored and the peritoneum resumes its normal state.

There is no evidence available to show that the reaction following drying may result in peritoneal adhesions. Even in the extreme degrees of desiccation, as seen in the experimental laboratory, this does not occur. This is due probably to the fact that the fluid first poured out on the surface after the intestines are returned to the abdomen does not produce the fibrillar fibrin necessary to the formation of adhesions.

The ordinary technic of the operating room is not always in accord with the scientific requirements. The operator endeavors to prevent cooling of the surface as well as drying. Since evaporation is believed to be the chief factor in the cooling of the surface he seeks to achieve both ends by the use of warm moist packs. When a moist pack is used, the cooling of the peritoneal surface is increased if care is not taken to prevent evaporation of the fluid escaping from the pack.

The exposure of the peritoneum to a moist pack is not without influence. The cells are united with each other by a cement substance. If a surface is exposed to a moist pack for even a few minutes this substance is in part at least removed. This is shown by the fact that silver no longer produces so marked a precipitate in the intracellular lines as it does on the normal peritoneum. Surfaces so affected have lost in a measure their capacity of protection. In addition if the hand of the operator or the nurse is allowed to act as a thermometer a pack too hot is likely to be used. Packs registering a temperature of 150° F. are not infrequently employed. This is sufficient to cause an increased exudate from between the cells and thus markedly to lessen the rate of absorption after a brief primary increase as may readily be proved by experimentation. This may readily lessen the defensive factors of the peritoneum. Unless the surgeon is so equipped that every requirement of the wet pack can be met its use does not justify the confidence he places in it.

These factors make the question of the use of the dry pack quite apropos. There is no abstraction of "Kittsubstanz" when the dry pack is employed. The temperature is more easily retained with a dry

pack than with a wet one, because the dry pack retains the natural heat of the region while the moist one, by producing an excess of moisture, increases the evaporation, and hence the abstraction of heat. Desiccation is prevented quite as well by the dry as by the wet pack because evaporation is prevented. The objection inveighed against the dry pack is that it injures the endothelium mechanically. There are no experiments recorded which tend to justify this belief. As a matter of fact it does not occur as will be discussed below.

As a protective against spreading fluid, as in draining off an abscess the dry pack has the advantage of taking up more of the fluid. The moisture in the wet pack serves only to increase capillarity and because of this tends to conduct infective material to sterile regions of the abdomen.

There is but one pack preferable to the dry pack in the abdomen; that is the one that remains in the dressing closet. If that portion of the organ only is exposed, which is the object of operative attack, no pack of any sort is needed.

Reaction Due to Mechanical Injury.—How well the endothelium is adapted to perform its function as a protective covering is easily demonstrated in attempts to remove the endothelial cells mechanically. In order to secure isolated cells for the purpose of microscopic study considerable force must be employed to remove them. In studying the histology of the basement membrane I used many methods to remove mechanically the endothelial cells. They can not be removed *en masse* by mere rubbing with an object such as a finger armed with a piece of gauze. Even when a needle is drawn with considerable force across the surface of the peritoneum the cells in the track of the needle only are disturbed. The cells tear but do not loosen. By subjecting the surface of the endothelium to such an injury and afterwards silvering the specimen it is easy to see just how little the cells have been disturbed. It is only when reactive processes are added to mechanical insults that the cells loosen readily. It is possible, of course, to secure an abrasion by violent rubbing with a piece of gauze as has been recorded, but a great deal of energy must be applied. Certainly no manipulation with gauze within the range of operative technic is sufficient to dislodge the cells. Trauma even to this extent is not followed by re-

action other than formation of an exudate with prompt endothelization. Much more deleterious sources of trauma are the bite of the forceps and the traction with sharp-pronged hooks. With these instruments solution of continuity of the basement membrane may be produced which may be the source of permanent adhesions. The same condition is produced in greater degree when a vessel is injured which results in subperitoneal hematomata. The ligation of pedicles leaving the stump exposed is a source of still more mischief. This stump is capable of exciting uninjured surfaces, with which it may come in contact, to reaction.

When mechanical injury extends to a sufficient degree to cause death of tissue a reactive process follows. The dead tissue institutes the same sequence of symptoms that bacterial toxins produce. There is vascular dilatation and abundant round cell infiltration. By this process the destroyed tissue is separated from the viable just as a slough follows infection. This process may be studied in peritoneal surfaces crushed with forceps or when end vessels are ligated as in the tip of the omentum, etc.

Reactions Due to Variations in Temperature.—The peritoneum probably is very resistant to low temperature, certainly so if we may reason from analogy from the experiments in transplanting sections of blood vessels. Short of refrigeration it does not seem to suffer. Certainly the cooling of the operating room has no morphologic effect on the integrity of the endothelium. This discussion does not take into account what effect the cooling may have on the contained nerves and blood. These points will be discussed in another place. Heat does not affect the peritoneum until a degree of above 120° is reached. Much above this point the albumins coagulate and the cells are loosened from the basement membrane. If the temperature continues to ascend, the perivascular tissue becomes coagulated and the vessels are occluded and the entire thickness of the membrane is destroyed.

Reaction Due to Bacteria.—Broadly speaking, this is the one common cause of peritoneal inflammations. It is the presence of bacteria on the surface of the peritoneum, which by multiplying produce an ever-increasing irritation upon the peritoneum, that causes the reaction. Factors which favor the production of a suitable culture media enhance the development of the bacteria.

Associated conditions which tend to supply such nutriment are hemorrhages or exudates due to disease or foreign bodies. The peritoneal cavity, however, is unlike a test tube, for in the presence of injury defensive factors are set in motion. Leucocytic agencies combat the invading host. This defensive capacity of the peritoneum is the important one when bacteria are introduced into the unchanged peritoneum as is shown in animal experimentation. These factors are complicated by the relative virulence of the various species of bacteria and even more by the relative virulence of the bacteria of the same species. Yet another factor enters to complicate the factor of etiology. Often preexisting reactive processes may prepare the peritoneum for invasion so that when bacteria do gain access to the surface of the peritoneum they are at once destroyed.

Such varied factors make the study of the bacterial etiology of peritonitis exceedingly complicated when discussed in the abstract, and it is only by the intensive study of concrete cases that the student appreciates the general rules underlying the problem.

Historical.—Wegner was the first to study the etiology of peritonitis in a comprehensive way. He demonstrated that the peritoneum absorbed with impunity considerable quantities of putrefying material. He also noted that such material in sufficient amount could be introduced that death by intoxication might result before the defensive functions of the peritoneum could be mobilized. If for any reason absorption of nonfatal doses was delayed, time might be had for the bacteria to multiply and a peritoneum thus became the site of a rapidly developing colony. Here we find clearly set forth several fundamental factors; the possibility of death by absorption of toxins before the reactive factors could be set into action, that is, before a peritonitis could develop; that small doses of bacteria might be destroyed before they could do harm; also that stagnating fluids in the peritoneal cavity would favor the development of the bacteria.

Much research followed that tended to obscure rather than to clarify the real problem. Chauveau repeated Wegner's experiments using micrococcus septicus puerperalis (probably streptococci) and determined that animals died from absorption of the septic products without obvious changes in the peritoneum. Grawitz

presented another phase of Wegner's work. He noted that when bacteria were introduced suspended in a fluid that was absorbed within a few hours infection did not follow, but if the fluid stagnated peritonitis resulted. According to him a phlegmon of the abdominal wall, as that arising in the needle tract through which the injection had been made, was necessary to provoke a peritonitis. Grawitz evidently was dealing with a strain of organisms of very much attenuated virulence for in repeating his researches a host of investigators did not find the staphylococcus so innoxious as the findings of Grawitz indicated. Thus Pawlowsky found relatively small amounts of staphylococcus aureus cultures to be fatal. This difference in results as demonstrated by Burginski showed that strains that were as innoxious as those used by Grawitz became markedly more malignant when passed once through an animal.

Baumgarten and A. Fraenkel evidently worked with weak strains while Orth, Rinné, Waterhouse, Walthard, Halsted, Tavel and Lanz, Silberschmidt, and Wieland worked with relatively more virulent cultures. Noetzel, working with Streptococcus pyogenes, proteus vulgaris, Bacillus coli, Bacillus pyocyaneus concluded that inflammation could be produced in the intact peritoneum if the strain of bacteria was virulent enough.

The alleged site of infection relative to the area of maximum absorption was discussed. Thus Beck digressed by speculating on the region where absorption might occur. He concluded that septic absorption could take place only through the diaphragm.

Clinical observation has abundantly proved the conclusions of the experimental laboratory. The amount of infectious material, the kind of bacteria, and the state of preparedness of the peritoneum are the dominating factors.

It may seem that a discussion of the reaction produced by specific types of bacteria would be the most scientific way in which to attack the problem. In experimental work such a discussion is possible, but in practice the varieties of bacteria active in a given case are often so numerous as to make such a scheme of limited application. For practical purposes a pathologic anatomic rather than a specific bacterial study is preferable. Nevertheless it is profitable to sum up the available knowledge of a distinctly bac-

teriologic study. This is of some value since in some instances pure cultures are encountered while in others some variety of bacteria may preponderate to such a degree that a bacteriologic and pathologic consideration harmonize to a great degree.

If one attempts to repeat on animals the researches that have been recorded relative to the pathogenicity of the various bacteria one secures data no less bewildering than that recorded in the literature. Animals with normal peritoneums absorb quantities of bacterial cultures that are astonishing. They die either from shock or intoxication, or they recover without the development of peritonitis. To secure specimens of bacterial peritonitis uncontaminated by foreign bodies is one of the most difficult feats I have encountered in animal experimentation.

From these studies it seems that so long as there is active absorption from the peritoneal cavity the animal is immune unless sufficient toxin is introduced with the culture to produce early death. This is true only when cultures are used suspended in an isotonic fluid. When bacteria are obtained from a localized abscess and used directly without first growing them on artificial media peritonitis is produced; the less the dilution the more certain the result. The reason for this seems to be that in order to secure the absorption of a hypertonic solution an exudate must form and by this exudation a culture medium is produced, for once the prompt absorption of the infective fluid is interfered with, the entire problem changes. The essential factor for the production of a peritonitis seems to be that bacteria shall remain in the peritoneal cavity for a certain time and it makes little difference whether bacteria are introduced with substances of themselves not capable of absorption, or substances themselves capable of exciting an exudate. Unfortunately a borderline between these two factors is hard to draw, since likely any substance incapable of absorption excites an effusion into the peritoneal cavity. For instance an agar slant, bearing on its surface an abundant growth, excites but little peritoneal exudate, yet a peritonitis will follow, when a like growth scraped from the agar slant and suspended in salt solution and so introduced will be absorbed with impunity.

There is a baffling variation in results with exudate producing substances. For instance, a bouillon culture of colon bacilli sus-

pended in salt solution is promptly absorbed. The same solution to which sterilized feces has been added produces a peritonitis. If the same amount of culture is meshed in a pledget of cotton and introduced, prompt encapsulation follows.

An attempt to determine the difference in the exudate produced by sterile feces and sterile cotton without the presence of bacteria gave little evidence. In that responding to feces, the exudate is less viscid and volume for volume reduces less silver nitrate. When exposed to the air the former deposits a granular debris while the latter produces fibrinous strands.

Antiseptic solution used in the peritoneal cavity, notably bichloride of mercury and iodine produce an exudate in which bacteria grow splendidly. This is true when the exudate is allowed to form before the bacteria are introduced. Even when the two are introduced simultaneously peritonitis is more apt to develop than when bacteria are introduced alone. In other words the irritation from the antiseptic drug aids the growth of bacteria more than its antiseptic power hinders them.

In general it may be said that the changes in the peritoneum are in the main similar for all varieties of bacteria. The factors common to all may first be considered. There are unmistakable variations, but these deviations may best be considered after the main changes have been discussed.

Because of the difficulties in producing a pure experimental peritonitis it is much more difficult to secure a uniform picture than after chemical irritation. The following account therefore is written from material secured at autopsy of the animal, the milder degree of reaction being secured from very early cases or at some distance from later ones.

A picture may be secured in animals, comparable to ulcer perforation in the human subject, by ligating a tip of gut wall and then closing the animal. In from one to four days the portion of gut so constricted becomes necrotic and the intestinal contents escape. Very frequently, however, adjacent coils of the intestine become adherent about the constricted portion of the gut and one secures at most a circumscribed abscess.

It will be necessary to consider the difference in the causes of the inflammation. A peritoneum irritated from an infection traveling

along its surface shows quite a different picture from one which has been approached from the depth of the tissues beneath.

In order to examine the data more minutely, it will be advantageous to examine separately the changes in the endothelium, those of the vessels, the perivascular connective tissue, and serous exudates.

The Changes in the Endothelial Cells.—The earliest change in the endothelial layer is a broadening of the intercellular lines as demonstrated by silver nitrate solutions (Fig. 142). This broadening is due to the exudation between the cells overflowing the edge of the cells, as can be demonstrated by making cross sections. This exudate represents the first act in the reactive process. In extremely toxic conditions this state does not occur.

The changes in the cell forms are dependent on the character of the irritative factor. In an average reaction as produced experimentally with gauze or as seen in subacute appendicitis the cell protoplasm becomes increased and the nuclei become thicker and more intensely staining (Fig. 143). The degree of such changes is very variable. In extreme cases the cells become cuboidal and even columnar (Fig. 144). When the cells show this form the first impression is naturally that they are new cells which have come to replace cells thrown off. This impression is heightened by the fact that in many cases endothelial cells are found in the exudate above (Fig. 144). In some instances, however, a gradual transition from the normal to the cuboidal can be observed, thus warranting the belief that the endothelial cells may actually undergo these astonishing changes (Fig. 145). This is a matter of considerable importance in the interpretation of certain pathologic conditions. It is equally interesting to determine whether or not such cells can return to the normal. We are warranted in saying that they can do so, for cells lying in the basement layer likewise swell and most certainly return to the normal when the irritation ceases.

Comparison with the cells lying in the subserosa is of even more importance when we come to consider the cause of this change in form. None of these do so except in the environment of fibrin. Unless an irritation is great enough to produce an exudate capable of forming fibrin, the changes do not occur. Once fibrin forms, the cells swell. It is of interest to note that diapedesis does not begin

Fig. 142.—The intercellular lines are broadened. Lugol's solution. Nitrate of silver. Oc. 2, Obj. 1/8.

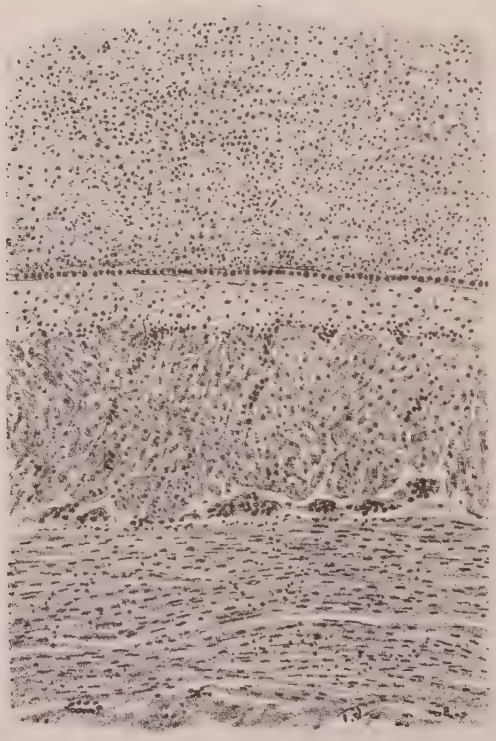

Fig. 143.—Acute inflammation of the peritoneum of the small intestine. The endothelial cell layer appears as a distinct row because of their large distinctly staining nuclei. Above this layer is the inflammatory exudate.

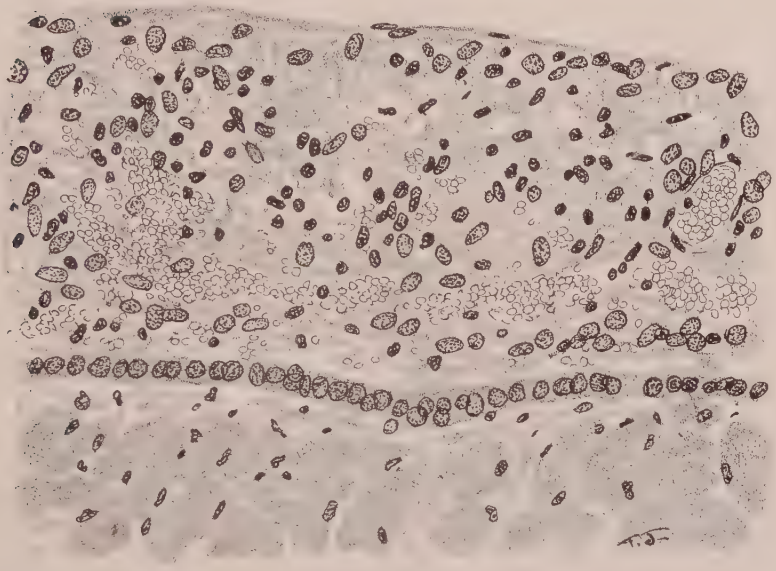

Fig. 144.—Same organ as in preceding cut but the inflammation is less acute. The endothelial cell layer is retained.

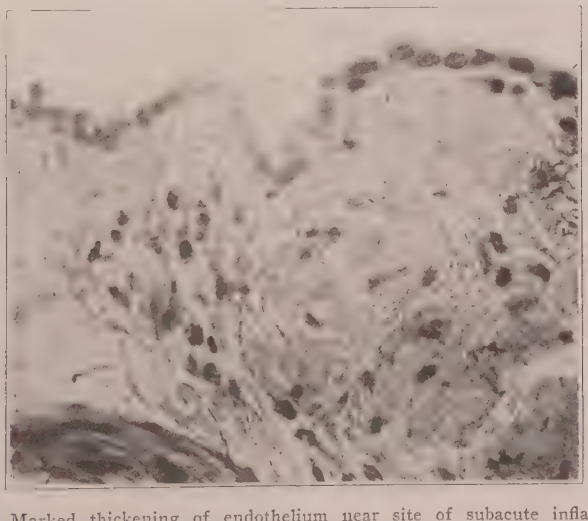

Fig. 145.—Marked thickening of endothelium near site of subacute inflammation.

until fibrin forms about the vessel. Most likely the act of diapedesis is the act of following along a fibrin bundle just as has often been described in tissue cultures and which was

described in the section on wound healing. What it is in these fibrin bundles that excites these changes in the cell is not known. The importance of this observation in establishing a like capacity in endothelial cell and so-called fixed tissue cell consists in the fact that it shows that the endothelial cells are closely related to the connective tissue cell. This fact makes it easier to believe that the endothelial cells when destroyed are replaced by the fixed tissue cells they so closely resemble in form and formation.

One of the most interesting features of this cell study is to note the close resemblance of endothelial and fixed tissue cell when irritated as compared to their appearance in the resting state. This difference is due largely to accessibility for study. When the basement membrane is teased and cells are isolated the cells resemble each other more closely. The difference is still striking enough to impress one with their adaptability.

This adaptability may be noted in chronic indurative processes as one sees sometimes about chronic appendices and elsewhere. Here parallel bundles of fibrin are seen lined by these endothelial cells. They appear as if each were capable of forming an endothelial layer, so much do they resemble the peritoneal endothelium in form and tinctorial reaction.

Endothelium once excited to reaction remains so until the fibrin surrounding it changes into fibrous tissue or is absorbed. It is interesting in this connection to note that epithelial cells can not advance over a surface after the fibrin has been fully formed as in the healing of an epidermal ulcer unless there are fibrin bundles for the epithelial cells to glide along. This relation of cells and fibrin is of vast importance as relates to the formation of malignant tumors.

When the peritoneal infection is malignant the changes above noted do not take place in the endothelial cells. They lose all capacity of staining and may be found as desquamated masses floating in the exudate.

Changes in the Circulation.—In the study of vascular changes in beginning inflammation, as observed in the animal mesentery, we tread in one of the earliest fields of experimental pathology. One would be rash indeed did he not approach the study of it with a feeling akin to reverence. My own studies in this field have been

rather to compare the studies of the old masters with special references to the relation diapedesis bears to the potential vessels already described and to the formation of perivascular fibrin.

While the classical experiments of Cohnheim were made on the web of a frog's foot, my own studies were made on the mesenteries and omenta of warm-blooded animals. Direct observations of the living tissue were made, but these were supplemented by the study of inflammation in the various stages of reaction.

When an exposed loop of gut with its mesentery is placed upon a slide, the following phenomena may be observed; in a varying period of time, depending on the age of the animals and the atmospheric conditions, the circulation in the vessels becomes more rapid. Since ordinarily several hours are required for these changes to begin, the advent of them can be expedited by the application of an irritant such as cantharides or iodine. The striking feature is that areas in which vessels previously were invisible now are seen to be traversed by numerous capillary vessels through which red cells rapidly pass (Fig. 146). When a chemical irritant is employed the red cells soon escape from the vessels into the surrounding tissue thus obscuring most of the changes except that of the dilatation of the vessels. The older authors believed these vessels to be newly formed. I have been able to demonstrate that the channels are always present requiring only the stimulus of the reactive process to cause them to begin functionating.

As the vessels become dilated and the blood stream becomes more rapid, the leucocytes come to lie in the peripheral portion of the current. This position of the leucocytes has been ascribed to their lighter specific gravity. That they are lighter was a pure assumption to fit the theory. The use of the centrifuge has disproved this theory. It is my opinion that this position of the leucocytes is due to the same influence which draws them toward the site of injury after they escape from the vessels. The endothelium lining the blood vessels swells in a manner similar to that described above for the endothelial cells. The actual increase in speed is not sufficient to throw them out, for it occurs when there is no perceptible increase in speed. If strong turpentine in oil is applied to the peritoneal surface, the blood stream becomes at once slowed and the peripheral stampede and the extravasation is more rapid than be-

fore. It has never been my fortune to actually see leucocytes escaping from a vessel. It is difficult to determine the relation of cells and vessel walls when observing such tissue. However, they collect about the peripheries of the capillaries and veins and it is fair to assume that they came from the lumen of these vessels. In stained sections it appears as if the protoplasmic masses which go to form the potential vessels become continuous with fibrin bundles in their immediate environment and that cells glide along these. In other words, the leucocytes escape from the po-

Fig. 146.—Potential vessels in beginning inflammation. The escape of leucocytes is just beginning.

tential vessels (Fig. 147). Cells seen about vessels in inflamed tissue before diapedesis begins are made up largely of fixed tissue cells which, like those of the endothelial cells already referred to, become swollen. Those that escape from the vessels early are largely polynuclear.

After the circulation has been accelerated for a time and the exudate about the vessels has attained a certain degree of completeness or at once if the irritant be powerful the circulation begins to slow. There is no obvious hindrance to the circulation,

however, and the retardation must be regarded as due to chemical changes in the environment rather than to a narrowing of the lumen of the vessels. This change is likely akin to the changes suffered by the peritoneal endothelium above noted. The cells are thicker, it is true, but this does not materially narrow the lumen. The silver-reducing power of the intercellular substance is increased and the coagulation time of the blood in these vessels is reduced.

Actual change in the intima can not be demonstrated except in

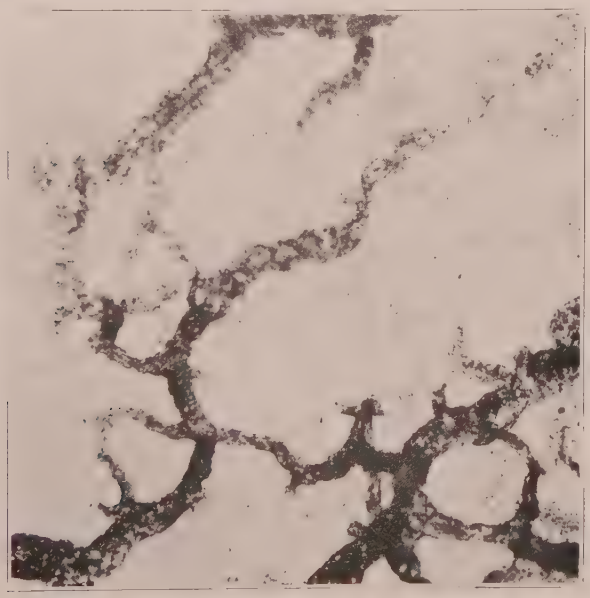

Fig. 147.—The much dilated potential vessels are surrounded by leucocytes.

the so-called hemorrhagic inflammation. This is not observed in human material except in certain violent irritations as after the perforation of an ulcer or near a necrotic or thrombotic process. In these instances there is an actual destruction and desquamation of the endothelial cells of the small capillaries. This can be determined by causing a hemorrhagic inflammation with turpentine, washing out the circulation with water and then injecting the circulation with silver nitrate. In such cases the vessels are

filled with blood, usually stagnant, and in many places the interstices are filled with red corpuscles (Fig. 148). This represents a very toxic type of inflammation.

At this stage we may picture the vessels dilated, the current slowed, with serous and leucocytic infiltration of the perivascular tissue. All these changes may take place in an hour or two under chemical stimulation, but under clinical conditions, require from twelve to twenty-four hours.

Changes of Perivascular Connective Tissue.—With the advent of the serous exudate, important changes take place in the connective tissue about the vessels. These changes involve the fixed tissue cells, the connective tissue fibrils and the intercellular substance. The changes in the fixed or wandering cells as the case

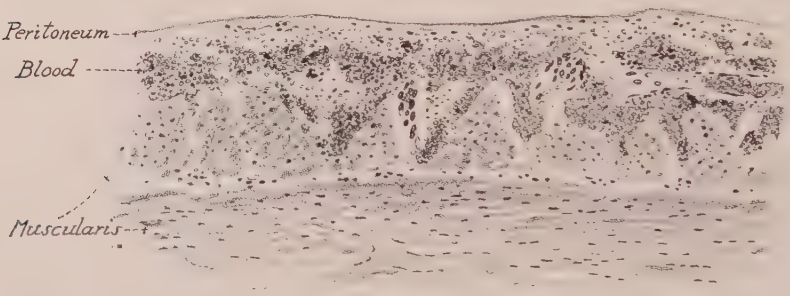

Fig. 148.—Acute hemorrhagic peritonitis. The subperitoneal layer is infiltrated with red cells.

may be have already been referred to. It may be recalled here that these cells are more abundant about the walls of the mesentery and omentum than in other tissues. These cells under the stimulus become globular and more conspicuous because of the increase in the protoplasmic mass and the more intense staining of their nuclei (Fig. 149). The changes in the cells make the degree of extravasation appear greater than it really is. The changes in the connective tissue are even more striking. The fibers swell and are separated by the exudate.

Tissues that normally stain with acid dyes refuse them. For instance, tissues, instead of staining red with fuchsin, take the yellow of the picric acid. That is, the tissue regresses to a point at which it existed before complete development, a stage nearer

fibrin. It does not revert so completely to the fibrin stages as to take the Weigert stain, however. This change in the tinctorial reaction occurs simultaneous with the changes in the cells above mentioned. Before either of the above changes occurs, there is an increase in the intercellular substance. The fibers are separated into minute fibrils so that the network becomes more complex. This exudate becomes precipitated into fibrin, at least when fixed, and seems to be the earliest response to irritation and

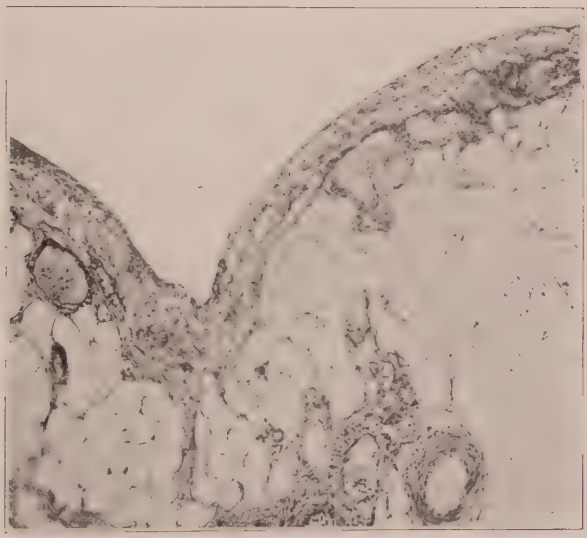

Fig. 149.—Perivascular endothelium has become thickened as the result of irritation from the great omentum in beginning appendicitis.

in certain slow sclerotic processes the reactive process likely does not proceed beyond this stage.

Now the leucocytes escape and their mode of migration seems to be along these fibrin bundles and only from vessels devoid of any wall save that of a layer of cells.

When this stage has existed for some time the tissue loses its resistance to mechanical forces as most operators who have pushed their fingers through a gut, the subject of a subacute inflammation, will recall. This attests strongly to the fibrinoid state of the connective tissue and is quite as impressive as the tinctorial

study. It may be added that did such changes not occur abscesses likely would never rupture spontaneously.

When the infective forces have been overcome many mononuclear leucocytes appear. These are mostly of the large variety with oval-shaped nuclei though many small ones with homogeneous deeply staining nuclei may be present. The relation of these cells to the changes in the tinctorial reaction of the connective tissue is problematic. The leucocytes use fibrin bundles apparently to facilitate their migration. The large mononuclears appear only after the height of the infection has been controlled by the leucocytes or in inflammations mild from the beginning.

What processes are at work which bring about the series of phenomena above described is difficult to say. Only irritants of a certain character produce it. Violent infections as by streptococci prevent this change. Gonococci produce the most fibrin and excite the cell exudation to the greatest degree. There must be some association with fibrin-producing substance and cell increase; very virulent infections prevent both.

The Formation of Serous Exudates.—Since the study of the inflammation of serous membranes began it has been noted that an exudate often forms which obscures the surface. It was found that this was due to a replacement of the serous covering with a fibrinous material. A discussion was waged as to whether this formed above or below the serosa it displaced.

Broadly speaking, two general opinions were held as to the source of this layer. The one group of investigators believed that it was derived from the fibrinoid degeneration of the subperitoneal tissue. The other believed that an exudate collected on the surface of the peritoneum and there coagulated and formed the membrane. A brief presentation of the argument employed will aid in giving a clearer picture of the changes which occur in the tissues.

Rokitansky compared it to a vegetation, and Virchow declared the fibrinous deposit to be nothing more than a degenerated and swollen tissue. Neumann is the chief advocate of the first view. He made exhaustive studies and came to the conclusion that in chronic cases the fibrinous masses were the result of the degeneration of connective tissue. In tuberculous materials he could

trace the change in all its stages. For acute cases Neumann admitted that the membrane was the product of an exudate. Langhans came to similar conclusions. The former, it is worthy of note, studied a periorchitis and a pleurisy and the latter a subacute hygroma. Opel in a study of twenty-nine postmortems found a degeneration of the connective tissue in the chronic cases but a fibrinous exudate in the acute cases since he found the endothelium unbroken under the fibrin. Grawitz taking a middle ground believed that the cells found in exudates are derived from connective tissue and that the protoplasm from which they are formed may produce fibrin. In other words, the fibrin is the result of degeneration of the intercellular substance. He believed that the entire thickness of the gut wall could undergo such a change. Marchand, in a rabid attack on Grawitz's views held that the change deep in the tissue is merely a production of fibrin. Sulton noted that in some exudates there is a layer of cells above and one below the exudate. He regards the layer on top of the exudate as the original endothelial layer while the lower he regards as an attempt at replacement of the cells over the basement membrane. Gaylord presents a clear discussion of the fibrinous exudate which forms on the surface but neglects the changes taking place in the connective tissue.

This confusion of opinion arose because the various observers studied different tissues and different processes. Those who studied acute exudative lesions and lesions experimentally produced found the mass above the basement membrane. Those who studied the chronic processes, particularly those lesions arising from some irritation within the subperitoneal tissue, found the lesions below the basement membrane. They correctly noted the change in the connective tissue and designated it fibrinoid degeneration.

The problem can best be understood by studying an acute inflammation (Fig. 150). Here there is an exudate both above and below the endothelial layer. That above the endothelium is made up of fibrin intermingled with polynuclear leucocytes and that below (*c*) is formed by fibrin deposited within the subperitoneal tissue. Here there is fibrin formation both above and below the endothelial layer. It is easy to understand that should the en-

dothelial layer become destroyed, the topography would be lost (Fig. 151). In such a specimen the upper part of the exudate (*a*) is fibrin exuded over the peritoneal endothelium (*b*) while the lower is made up of fibrinoid degeneration of the subperitoneal connective (*c*) tissue together with some new fibrin deposited within its meshes.

That the exudate is above the basement membrane in the acute cases can be easily proved by placing a foreign body in the peritoneal cavity or by injecting weak turpentine emulsions. An exudate quickly forms on the surface of the peritoneum which reacts to the fibrin dyes. Often the endothelial cells remain on a

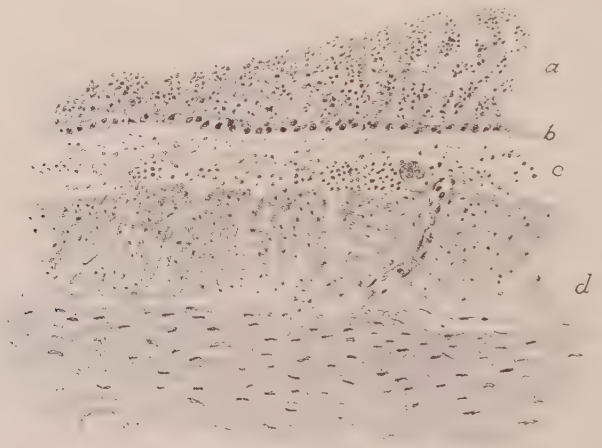

Fig. 150.—Early fibrin exudation. The endothelial layer is but little thickened.

little changed basement membrane to mark the normal tissue outlines. There may be no change in the subperitoneal tissues at all in this stage, but in human material there is nearly always some edema or fibrinous exudate in the subperitoneal tissue, usually the greater the longer the process has continued.

In the slowly progressing cases there are two possibilities which may cause confusion. The membrane may primarily be the result of an exudate as in the experiment above noted. This at first is fibrinous in character but later develops into fibers and secures a layer of endothelium on its surface. If observed at this time the new endothelial layer may be mistaken for the original

one and it may appear as if the whole process had been played below the endothelial layer (Fig. 152). This is more often noted

Fig. 151.—More advanced inflammation than in the preceding figure. The endothelial layer (*b*) is difficult to trace.

in cases of chronic pleurisy than in peritonitis, because the infections in the pleural cavity are less violent and hence more readily

organize. This layer of tissue formed above the basement membrane may secure an independent set of vessels just as has been noted in development of pseudomembranes covering foreign bodies and then it is distinguished with difficulty from the original peritoneum. In such specimens the original layer of endothelial cells appears as a second more deeply lying layer. This is the process which takes place when an adhesion or pseudomembrane is produced. This is noted most often in subacute salpingitis of gonococcic origin.

This organization of the exudate is the exception even in

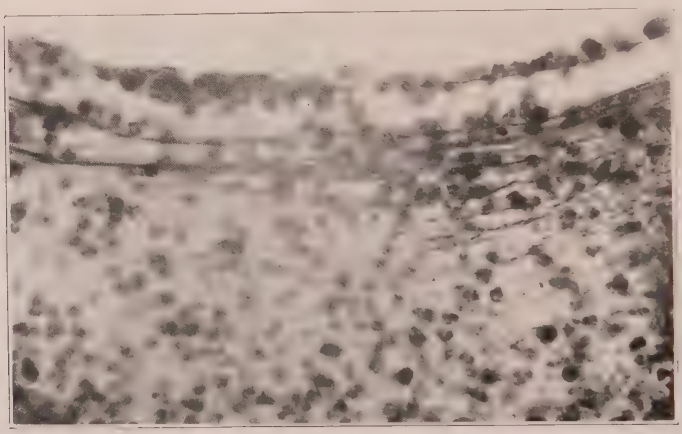

Fig. 152.—Superficial fibrin layer of peritoneal exudate with endothelioid cells on the surface.

chronic processes. Usually the entire exudate is absorbed leaving an endothelial layer on the original basement membrane.

In the chronic type the picture is chiefly played beneath the basement membrane. Here the infiltration within the tissues produces a change in the connective tissue correctly referred to as fibrinoid in character. The fibers of the limiting membrane and the subperitoneal tissue are so separated by the exudate that their topography is much confused and once the endothelial layer is lost the surface line may be located with difficulty. One can follow the process accurately only by means of the fibrin dyes. Mallory's stain renders the greatest aid here since it identifies the fibrin fibrils after they no longer accept the fuchsin in Van Gieson's

stain. By this means one is able to differentiate between these and the fibrin fibrils produced within the meshes of this tissue as well as that forming in the exudate above the endothelial layer. It is only very late in the process that the connective tissue bundles undergo so great a degeneration that they do not stain specifically. At this stage the subperitoneal line may often be identified by the presence of thick-walled vessels.

Ziegler believes the change which the connective tissue undergoes is a hyaline degeneration. Against this assumption is both the histochemistry of the fibers and the fact that they readily revert to the normal when the process ceases. It represents a metamorphosis and not a degeneration.

Though the tinctorial reaction of the interstitial fibrinoid fibrins is similar to that noted in the fibrillar fibrin in wound healing, there is an essential physiologic difference in that they do not tend to form new connective tissue. The importance of this observation is that such an organ may remain thick for many months, yet when the causative factor is overcome the thickened wall returns to its normal dimensions, which obviously could not be the case if a production of fibrous tissue had taken place.

It is interesting to speculate on the purpose of this fibrinoid degeneration. When in this state the fibers are thicker and of course longer to accommodate the increased size of the organ. This may be noted for instance in a swollen appendix. When this organ is as large as a thumb the subperitoneal fibrous tissue must of course be enormously lengthened to meet this increase in circumference. When the swelling subsides this tissue resumes its normal state. One observes a similar condition when a wound heals. The original fibers are long but contract much as the scar increases in age. This type of degeneration takes place only when the tissue is waging a more or less successful conflict with the invading infection. What part this may have in the reactive process is difficult to say. It does seem, judging purely from sequence and association, that their degeneration is a prime necessity for the migration of the large mononuclears.

The most frequent site for this type of change is the appendix and contiguous guts and in salpingitis. These organs may be

enormously thickened and remain so for many months yet restoration to normal is certain if the focus of irritation is removed. During my more timid years as a surgical practitioner I frequently noted appendices as thick as my finger at the time of the drainage of an abscess. At the second operation the organ would be found to be of normal size. The restoration often was so complete as a matter of fact that had the source of the appendix so removed been unknown one could hardly say from the microscopic appearance of the organ that it had ever been the site of inflamma-

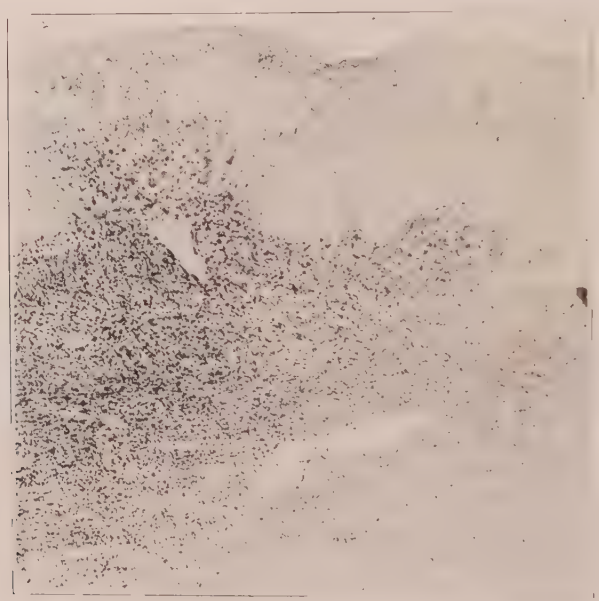

Fig. 153.—Intense peritonitis showing amorphous exudate in the upper part and cellular exudate in the lower part of the figure.

tion. Those who have operated on cases of salpingitis in the acute and chronic stages will require no further argumentation.

Pertaining to the cells in subperitoneal exudates it can only be said that the cell content depends on the character and age of the process. In toxic cases, as in streptococci infection, the exudate may be composed largely of granular material with exceedingly few cells, polynuclears predominating early in the process (Fig. 153), with a predominance of mononuclears later in the disease

after fibrinoid degeneration of the connective tissue has taken place. The accumulation of these mononuclears may be so pronounced that associated with a vastly thickened gut wall the condition may readily be mistaken for a sarcoma. No doubt this mistake is often made, as a perusal of the literature will suggest. The differential diagnosis will be discussed in the chapter on tumors.

With long standing inflammation a proliferation of fibers with

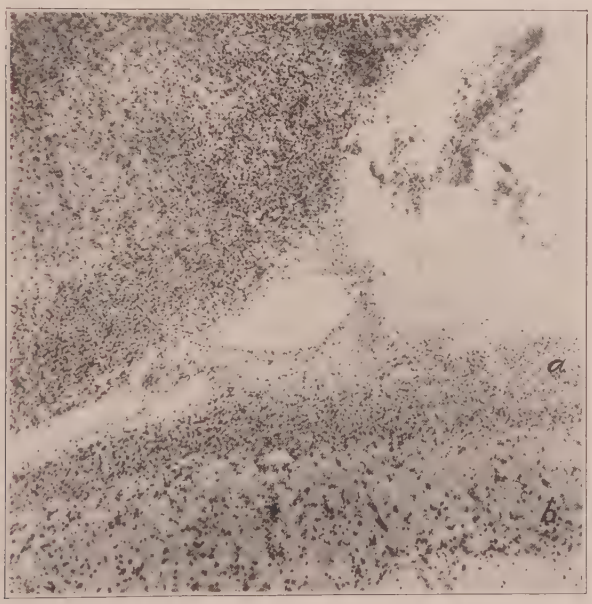

Fig. 154.—Acute peritonitis. The peritoneum (*a*) is lifted from the gut wall by a hemorrhagic exudate (*b*).

a thickening of the fibrinous layer may take place. This occurs not infrequently to a slight degree as may be noted after repeated attacks of appendicitis in which the muscular layer is decreased and the fibrous layer is thickened. When there is an actual necrosis of connective tissue a new fibrous tissue takes its place and may then form in excess. There seems to be a distinct difference in so far as the stimulus to fibrous tissue goes between actual tissue necrosis and irritation from infection. In the former the fibrin proceeds without delay to the formation of fibrous tissue

while in the latter the fibrinoid state may remain stationary for many months.

The foregoing account of the formation of the exudate applies to surfaces capable of controlling the irritation. When the irritation overwhelms the tissues the picture is quite another one. Here the connective tissue refuses all stains and the exudate on the surface is composed of granular debris, devoid of any cellular content or in the less acute many polynuclear cells representing various stages of degeneration may be found. In yet more slowly developing processes here and there a solitary fibrin bundle may be observed. There is the picture of acute spreading peritonitis elevating the basement membrane some distance from the muscle layer (Fig. 154) and obscuring all the blood vessels.

Bibliography

ADAMI: Inflammation, Macmillan Co., London, 1899.

APEL: Ueber die Herkunft des Fibrins auf serösen Häuten, Diss, Göttingen, 1895,

BAUMGARTEN: Lehrbuch d. pathologischen Mykologie, 1890, p. 417.

BECK: Ueber die Aufsaugung fein verteilter Körper aus den serösen Höhlen, Wien. klin. Wchnschr., 1893, vi, 823.

BURGINSKI: Ueber die pathogene Wirkung des Staphylococcus aureus auf einige Tiere, Arb. a. d. Geb. d. path. Anat. u. Bact. Inst. zu Tübingen, Braunschweig., 1891, i, 63.

CHAUVEAU: Sur septicémie peurperale experimentale, Lyon méd., 1882, xli, 272.

FRAENKEL, A.: Die puerperale Peritonitis, Deutsch. med. Wchnschr., 1884, x, 212.

GAYLORD: Fibrinous Exudates and Fibrinoid Degeneration, Jour. Exper. Med., 1908, iii, 1.

GRAWITZ: Statistischer und experimenteller pathologischer Beitrag zur Kenntnis der Peritonitis, Charité-Ann., 1886, xi, 770.

Atlas der path. Geweblehre, Berlin, 1893.

HALSTED: The Treatment of Wounds with Especial Reference to the Value of the Blood Clot in the Management of Clear Spaces, Johns Hopkins Hosp. Rep., 1891, ii, 255.

LANGHANS: Deutsch. Chir., Lief, l, 1887.

MARCHAND: Der Process der Wundheilung, Enke, Stuttgart, 1901.

Zur Kenntnis der fibrinösen Exudate bei Entzündungen, Virchows Arch. f. path. Anat., 1896, cxlv, 279.

NEUMANN: Zur Kenntnis der fibrinoiden Degeneration des Bindegewebes bei Entzündung, Virchows Arch. f. path. Anat., 1896, cxliv, 201; and ibid., Fibrinoide Degeneration und fibrinöse Exudation, 1896, cxliv, 201.

NOETZEL: Ueber peritoneale Resorption und Infektion, Arch.. f. klin. Chir., 1898, lvii, 311.

OPEL: Diss. Gütingen, 1895.

ORTH: Experimentelles über Peritonitis, 62 Verhandlung d. Naturforscher und Aerzte zu Heidelberg, Zentralbl. f. Chir., 1889, xvi, 849.

PAWLOWSKY: Beiträge zur Aetiologie und Enstehungsweise der akuten Peritonitis, Zentralbl. f. Chir., 1887, xiv, 881.

v. Recklinghausen: Erkrankungen des Kreislaufs und der Ernährung Entke, Stuttgart, 1878.

Rinné: Ueber den Eiterungsprozess und seine Metastasen, Arch. f. klin. Chir., 1889, xxxix, 1.

Rokitansky: Lehrb. a. path. Anat., Wien, 1855, ed. 3, i.

Silberschmidt: Experimentelle Untersuchungen über die bei der Entstehung der Perforationsperitonitis wirksamen Faktoren des Darminhaltes, Mitt. klin. u. Med. Inst. d. Schweiz, 1893, i, Rheike, 1″ Hefte.

Suchardt: Die Entstehung der subacuten Hygrome, Virchows Arch. f. path. Anat., 1890, cxxi, 305.

Sulton: Die feien Körper der Tunica vaginalis, Virchows Arch, f. path. Anat., 1895, cxl, 449.

Tavel and Lanz: Ueber die Aetiologie der Peritonitis, Mitt. klin. u. med. Inst. der Schweiz, 1893, i, Rheike, 1″ Hefte.

Virchow: Gesamte Abhandlungen, Frankfort, A. M., 1856, p. 136; and Virchows Arch. f. path. Anat., 1858, xv, 530.

Walthard: Experimenteller Beitiagen zur Kenntnis der aetiologische Studien Peritonitis, Kopenhagen, 1891.

Waterhouse: Experimentelle Untersuchungen über Peritonitis, Virchows Arch. f. path. Anat., 1890, cxix, 342.

Wegner: Chirurgische Bemerkungen über die Peritonealhöhle mit besonderer Berücksichtigung der Ovariotomie, Arch. f. klin. Chir., 1876, xx, 51.

Wieland: Experimentelle Untersuchungen über die Entstehung der circumscripten und diffusen Peritonitis, Mitt. klin. u. med. Inst. der Schweiz, 1893, ii, Rheie, 7″ Hefte.

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